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THE PLANT DISEASE REPORTER

Issued By

PLANT DISEASE EPIDEMICS
and
IDENTIFICATION SECTION

AGRICULTURAL RESEARCH SERVICE

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PLANT DISEASE EPIDEMICS AND IDENTIFICATION SECTION

Horticultural Crops Research Branch — Plant Industry Station, Beltsville, Maryland
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THE BÜCHNER FUNNEL AS AN AID IN COLLECTING AND CONCENTRATING NEMATODE POPULATIONS

William A. Feder and Julius Feldmesser

The Büchner funnel, a common tool of the chemist, is used as a filter. It is made of glass and contains a wafer-shaped insert of fritted glass of uniform porosity. (Fig. 1)[1]. In operation the funnel is seated in the neck of a side-arm flask and liquids to be filtered must be drawn through it by means of a vacuum line attached to the side-arm. The present note concerns this funnel as an adjunct to nematological techniques such as the use of the nested standard sieve series and various Baermann funnel modifications. It enables the handling of large concentrations of living and fixed nematodes in minimal quantities of liquids.

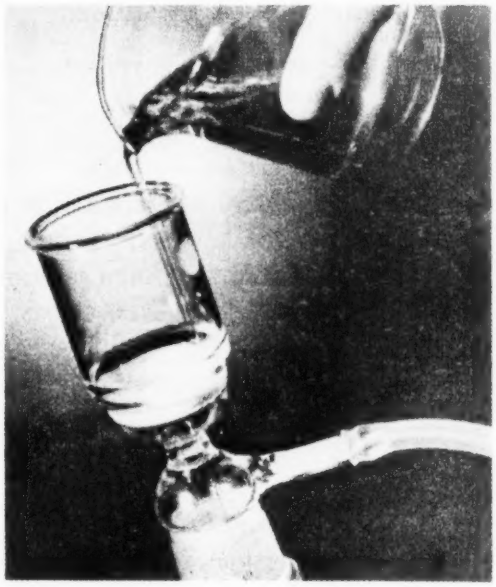

FIGURE 1. The Büchner funnel in use. The side arm (lower right) is attached to a vacuum line.

Various techniques involving sieves or Baermann funnels as means of collecting nematodes are described in the literature, the most pertinent being those of Linford (2) and of Christie and Perry (1). Linford's method involves the concentration of nematodes from infested material by pouring suspensions of plant debris into a small folded filter paper set in a stemless funnel. The funnel is then placed in a small beaker so that the tip of the filter paper is in contact with water. The nematodes migrate through the paper within 12 to 48 hours. The use of the Büchner funnel after this migration is complete allows greater concentration of the nematodes. The water suspension of nematodes is passed through the funnel. The nematodes caught on the surface of the fritted glass wafer are then washed several times with distilled water, which is drawn through the funnel. The funnel is then removed from the side-arm flask and all the nematodes are flushed off the surface of the wafer in as little as 5 cc of water.

Christie and Perry (1) wash nematode-infested soil through nested No. 24 and No. 200 standard mesh sieves. The material on the 200-mesh sieve is then introduced into a Baermann funnel modified by the addition of a muslin screen in contact with the water. The nematodes usually migrate through the muslin, within 12 to 24 hours, into the water and most of them sink into the area just above the stop-cock. During this 24-hour period, small amounts of the nematode suspension are drawn off at regular intervals. The use of the Büchner funnel makes periodic tapping of the Baermann funnel unnecessary. The nematode-bearing material is allowed to soak undisturbed for the maximum time period. The Baermann funnel is then drained

[1] Figure 1 courtesy of the Corning Glass Works, Corning, New York.

and all of the water is passed through the Büchner funnel. Nematodes caught on the surface of the fritted glass wafer can then be flushed off as in the previous process. In addition, the authors' experiences show that the 24-hour period, usually deemed long enough to allow for the complete migration of populations of large and vigorous nematodes such as Belonolaimus gracilis, is actually insufficient. It has been found that significantly large numbers of these nematodes remain unharvested in the funnel. In order to recover a maximum number of nematodes from these Baermann funnels, it is wise to drain the entire funnel and to proceed with the Büchner funnel as described above.

This tool is also of general value in the handling of soil and plant-free nematodes in suspension in fixatives, stains, wash solutions and toxicants.

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EXPERIMENTS WITH SYSTOX AS A SOIL DRENCH FOR CONTROL OF THE NEMATODE, MELOIDOGYNE INCOGNITA, OF TOMATOES

Max J. Fielding

According to Sasser et al. (3), larvae of the golden nematode of potatoes (Heterodera rostochiensis Wollenweber, 1923) were killed when exposed to several concentrations of Systox[1] in vitro, but were not killed when exposed in the soil, the material having been applied as a drench before planting. On the other hand, the same authors found that root-knot nematodes were controlled in greenhouse tests of tomato seedlings by soil drenches of Systox at concentrations as low as 0.03%, applied one week prior to planting; these seedling plants showed some chemical injury. In later experiments Sasser (4) controlled root-knot nematodes by applying Systox at various rates as a soil drench, before planting tomatoes or cucumbers or both. The most favorable dosage from the standpoint of control with little or no phytotoxicity was obtained with 300 ml. of a 0.1% solution per 5-inch pot (8.1 ml. of active ingredient per cubic foot of soil). Still later, Sasser (5) stated that his experiments to date had not indicated that Systox was effective as a systemic nematocide. Kuitert (2) attempted control of root-knot nematodes on gardenia plants with soil drenches of demeton (Systox) (41.8% emulsifiable) and reported: "Roots of the plants in the pots which were not treated generally remained in the original core and the failure of the roots to penetrate into the surrounding soil was attributed to the presence of nematodes. The demeton treatment could have controlled the nematodes directly, it may have been absorbed by the plant, thus making the plant resistant to the nematodes, or it may have had some nutritional effect."

A recent paper by Beale (1) reported that Kuitert and Tissot obtained control of root-knot nematodes on gardenias by drenching the soil with a solution of two ounces of Systex to 100 gallons of water.

Potted tomato plants which had been artificially infected with Meloidogyne incognita (Kofoid & White, 1919) Chitwood, 1949, one month before were treated with Systox by immersing the pot and the lower portion of the plant in a 2 1/2-gallon bucket containing the Systox solution for one hour. Concentrations of Systox were as follows:

Concentration	Percent active ingredient
1	.0075
2	.031
3	.062
4	.096
5	.2
6	Check

Four replicates were made for each treatment.

After this treatment the plants were placed in the greenhouse and allowed to grow for one month. All plants survived the treatment, but definite injury was noted at concentrations of .031% and above. The plants were then removed and the roots examined for larvae of the root-knot nematode. Active larvae were found in all treatments. Inoculum, from each of the tests, was then placed in 6-inch pots and young tomato seedlings grown in sterile soil were planted. These seedlings examined after one month were found to be severely infected with M. incognita. No significant difference could be found between results with inoculum from the various treated plants and with that from the checks. All plants including the checks had a moderate to heavy root-knot infection. It must be assumed from these tests that the root-knot nematode was not significantly reduced by the treatments. The writer agrees with Sasser (4) that Systox cannot be recommended for the control of root-knot nematodes.

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[1] An organic phosphate systemic insecticide which contains the following active ingredients: Diethoxy thiophosphoric acid ester of 2-(ethylmercapto)-ethanol 42.4%, related organic phosphates 5.7%; inert ingredients: emulsifier (Thiosolve 41-1A) 51.9%.

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STUNTING OF TOMATO ASSOCIATED WITH PRATYLENCHUS PENETRANS, AN APPARENT MIGRANT FROM AN ADJOINING PEACH ORCHARD[1]

W. B. Mountain and J. C. Fisher[2]

In studies of the root lesion nematode Pratylenchus in southwestern Ontario, three species have thus far been found. The most ubiquitous species appears to be Pratylenchus minyus Sher & Allen which infests the roots of a wide range of crops and is widespread in the lighter soils along the north shore of Lake Erie. A second species of Pratylenchus occurs on the roots of red clover and has not been described. Pratylenchus penetrans (Cobb) is the third species occurring in southwestern Ontario and has been observed in extensive numbers only in the roots of peach.

In the spring of 1954, a small field of tomatoes near Leamington, Ontario, was called to the attention of the Harrow Laboratory because of a rather peculiar growth pattern of the crop. The field, of Fox Sandy Loam, is rectangular with the long axis running north and south. When examined in late June, the tomato plants growing on the east side of this field were stunted, apparently having made little growth since they had been transplanted to the field. In contrast, the tomato plants on the west side of the field were large and growth had been vigorous. The line demarcating the healthy from the stunted plants ran north and south, approximately through the center of the field. To the east of this field and separated from it only by a laneway was a peach orchard approximately ten years old. The grower claimed that the stunting of tomato had been apparent for the past seven years, and that during this time the area of stunted plants had gradually increased away from the side adjacent to the peach orchard.

An examination of the plants showed that the stunting was associated with the condition of of the roots. Healthy plants from the west side of the field possessed well-developed, much-branched roots, practically free from lesions. On the other hand, the roots of the stunted plants were poorly developed, with very few branch roots. Many lesions were evident and most of the lateral roots had apparently died back from the tips. The average weight of the healthy root systems was more than four times as great as that of the affected root systems.

Tomato roots from both sides of the field were placed in Baermann funnels and the soil from around these roots was screened for nematodes. The affected roots from the east side of the field consistently contained more than ten times as many Pratylenchus per plant as the healthy roots from the west side of the field. No other parasitic nematodes were observed. The grower could offer no possible explanation for this uneven distribution of root lesion nematodes in the field.

However, in the course of counting nematodes, numerous Pratylenchus males were observed, which indicated that at least some of these specimens could not be Pratylenchus minyus, inasmuch as males of this species have never been observed in Ontario. Microscopic examination of specimens from the field in question showed both P. penetrans and P. minyus to be present, the former species being much more numerous than the latter. In view of the common association of Pratylenchus penetrans with peach roots, the question arose as to the possibility of migration of this nematode from the peach orchard immediately to the east of the tomato field. Accordingly, tomato plants were collected at varying distances from the peach orchard for examination; from the east edge, the center, and the west edge. Studies of the population of Pratylenchus were made from the roots and soil at each location. First, 100 Pratylenchus specimens from each location were selected at random, cleared and identified, to give the percentage occurrence of each species at each location. Then the total population of each species was calculated for each location. These figures are presented in Table 1.

It may be seen from Table 1 that whereas the population of Pratylenchus minyus was evenly distributed throughout the field, that of Pratylenchus penetrans showed an uneven distribution, being abundant on the side adjacent to the peach orchard and sparse across the field.

An examination of peach roots from the adjoining orchard and of the roots of weeds used as a cover crop in the orchard showed a high population of Pratylenchus penetrans; on the other hand Pratylenchus minyus could not be found.

It is postulated that the population of Pratylenchus penetrans was built up originally in the

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[1] Contribution No. 1418, from the Botany and Plant Pathology Division, Science Service, Canada Department of Agriculture, Ottawa, Ontario.

[2] Associate Plant Pathologist and Extension Officer, respectively.

Table 1. Populations of two species of Pratylenchus from different locations in a tomato field adjacent to a peach orchard.

Location of tomato plants	Pratylenchus from roots (per gram of root)		Pratylenchus from soil (per gram of soil)	
	P. minyus	P. penetrans	P. minyus	P. penetrans
East edge (nearest to orchard)	22	166	16	115
Center	17	40	22	52
West edge	23	5	12	3

peach orchard (possibly introduced in the nursery stock) and eventually migrated into the adjoining field, where at the time of this study, it formed the bulk of the population of Pratylenchus over the adjacent half of the tomato field. On the other hand, the evenly distributed, considerably lower population of Pratylenchus minyus might represent the normal parasitic nematode population of a typical tomato field in the area concerned.

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SOIL FUMIGATION FOR THE CONTROL OF NEMATODES ON GRAPE REPLANTS

D. J. Raski

The premature decline of vineyards accompanied by decreasing productivity and reduced vigor is a problem of major importance to the grape industry of California. Injury of this nature is known to be particularly common throughout the San Joaquin Valley and Southern California and has been observed in plantings of all ages varying from five-year-old to 60-year-old grapevines. In some cases the damage is confined to small, localized areas within a vineyard but in others may be extensive throughout large plantings in which most of the vines show some degree of injury.

Attempts to replant such vineyards after removing the weakened vines often result in failures of the plants to establish and thrive. In many respects this is a problem of greater importance than the weakening of old vineyards in that some level of production can usually be maintained in the older plantings whereas many of the new plantings never produce a crop.

Nematodes parasitic in and on the roots of vines probably represent one of the most important factors in the decline of old vineyards as well as the difficulties encountered in trying to establish replants. Yet there is relatively little information in the literature regarding these pests of grapes. One of the first records in this country was a description by Neal (4) of root-knot nematode on the roots of grapevines in Florida. Snyder (5) reported the development of rootstocks resistant to this nematode. More recently Lider (2) reported further studies on the inheritance of nematode resistance in Vitis spp. Machmer (3) found an improvement in the growth of grapevines planted in root-knot infested soil which had been treated with chloropicrin. Allen (1) summarized the plant parasitic nematode problem in California and characterized root-knot and root-lesion nematodes as the most important species attacking grapevines in this State.

Most of the collections of root-knot nematode on grapes in California have been identified as Meloidogyne incognita var. acrita, which is very widely distributed and is especially severe in the Fresno, Lodi, Delano, and Arvin districts. Another species, M. javanica, is also known to occur on grapes but has been collected in relatively few vineyards up to the present time. In general, the damage by root-knot nematode attacks tends to occur more frequently and is more intense on the light, sandy type soils.

The root-lesion nematode, Pratylenchus vulnus, appears to be an equally serious parasite wherever it has been found infecting the roots of grapes. It has been observed in soil and root samples from most of the important grape-producing counties but is not encountered nearly as frequently as the root-knot nematode. It is not unusual to find both of these species in the same vineyard. At least one other species of root-lesion nematode, P. minyus, has been found quite often in soils taken from about the roots of grapes but so far has not been definitely established as a parasite of these vines.

A number of other nematode species are very frequently found associated with declining vineyards, including Xiphinema americanum, X. index; Paratylenchus hamatus; Criconemoides xenoplax, C. mutabile; and Trichodorus spp. The ability of these species to injure grapevines has not been determined but further study must be directed toward these pests since they occur so commonly in the vineyards in California.

Soil fumigation tests directed toward the improvement of existing vines infected with nematodes have been unsuccessful largely because of the phytotoxic properties of the chemicals which have been available. The work reported here was started in the autumn of 1952 and was designed to compare the efficacy of various soil fumigants in helping to establish grape replants made in old vineyards infested with nematodes. Five experimental field plots established on three different varieties of grapes were arranged in cooperation with the University of California Agricultural Extension Service[1].

[1]Acknowledgments: The following Farm Advisors contributed valuable assistance to the progress of this work: Mr. F. Gordon Mitchell, San Joaquin County; Mr. Verner Carlson, Merced County; Mr. Paul Baranek, Madera County; Mr. A. Kasimatis, Kern County. Application of the fumigants was made by experimental machines belonging to the Shell Chemical Co. and the Dow Chemical Co. with the assistance of Mr. C. W. McBeth and Mr. H. Lembright of these two companies respectively. Dr. L. Lider of the Department of Viticulture, University of California, Davis, generously cooperated in rating the vines and evaluating their growth at the end of the first growing season.

The principal materials tested in all five plots were ethylene dibromide and 1, 3-dichloropropene; 1, 2-dichloropropane mixture (hereinafter referred to as EDB and D-D respectively). The formulation of EDB (66% by volume or 84% by weight) commonly used for commercial fumigation was used throughout these tests but all treatments reported here are expressed as the amounts of actual EDB that were applied. In two of the experiments OS-1897, a new experimental chemical supplied by the Shell Chemical Co., and 1, 3-chlorobromopropene (referred to as CBP-55) were included among the treatments.

Four of the experiments were designed with a single replication for each treatment. This was necessary because of the limited size of the plantings available for the tests. Since the success of the treatments would be measured over a period of years, it was decided that reinfestation from surrounding vines would be an important consideration in the outline of the experiments. Therefore, the plots were made as large as possible at the expense of replication.

The chemicals were injected by chisels at a depth of approximately 6 to 8 inches, and wherever equipment was available a harrow and/or roller were used to help seal the fumigants in the soil.

Soil samples were taken at the end of the first growing season to determine the nematode populations present at that time. A soil auger was used to secure samples at each 1-foot level down to a depth of 3 feet, except in Plot #5 which was sampled to a 2-foot depth. Root-knot nematode counts were made from galls on tomato seedlings grown in 200 cc. aliquots of soil from each sample.

The vines of each plot were pruned normally during the winter of 1953-54 and wherever possible individual weights were obtained from the vines of the center row (center two rows of Plot #4) of each treatment. In Plots #3 and #5 this was not possible and averages were obtained from composite weights of all the vines from the center row of each treatment.

Plot #1: One experiment was established on the property of Mr. Leon Handel near Lodi. The vineyard had been planted to Tokay grapes which were infected with M. incognita var. acrita. The vines were removed during the winter of 1949-50 and for three succeeding years the field was planted to bean varieties susceptible to the nematode. This resulted in a very high population of root-knot nematodes at the end of the 1952 growing season.

The soil type is a sandy loam with a moisture equivalent of 7.2%. The moisture content at the time of treatment was 7.0% with a soil temperature at the 6-inch depth of 7° C. These data apply to the D-D treatments, which were made on November 28, 1952. A heavy rain fell within 48 hours after the application. The EDB treatments were made February 11, 1953, and Tokay cuttings were planted March 25, 1953. Each treatment covered an area 4 rows and 21-22 vines long.

Average gall counts from soil samples taken on January 8, 1954, and the average weights of cane prunings made February 2, 1954, are tabulated in Table 1.

The nematode control in this plot was very effective, failing only in one treatment, EDB at 6 gal./A. This high count resulted from only two samples of the ten which were collected in this treatment. The average count for these two samples was 124 galls per pint of soil, whereas the average for the other eight samples was 2.8 galls. It is possible that an uneven application of the chemical could account for this apparently localized loss of control.

The response of the vines to the fumigants is greater than in any other of the experimental plots. The alternate planting of beans may have resulted in a much greater nematode population than is ordinarily found in vineyards, which could account for this extreme degree of injury to the new plantings in untreated soil. The control by fumigants, however, was better than in any of the other plots.

Plot #2: The property of Mr. J. L. Reuscher near Lodi was the site of another plot, involving Tokay grapes infected with M. incognita var. acrita which were pulled out during November 1952. The soil type was a sandy loam with a moisture equivalent of 8.0%. The moisture content at the time of treatment, February 11, 1953, was 7.6% and the soil temperature at the 6-inch depth was 8° C. Each treatment was 3 rows wide, 20 vines per row. The new planting of Tokay cuttings was made March 22, 1953.

Average gall counts from soil samples collected January 8, 1954, and average pruning weights taken February 2, 1954, are reported in Table 2. During 1953 the grower allowed a heavy weed growth to develop as a frost protection for the young vines.

The response to fumigation, as indicated by the comparative weights of cane prunings, is not so great as in Plot #1 possibly owing to a lower population of nematodes present at the time of treatment. The gall counts show a significant nematode survival in most of the treatments.

Table 1. Average cane growth and nematode counts in grape replant fumigation plot #1. Leon Handel property, Tokay variety, Lodi, California.

Treatment	Rate gal./A	Average cane weight gms. per vine	Average root-knot gall count per pint of soil
None		0.09	31.6
D-D	20	53.1	2.0
D-D	40	40.8	0.2
D-D	60	80.4	0.4
EDB	6	45.7	27.0
EDB	9	111.5	0.7

Table 2. Average cane growth and nematode counts in grape replant fumigation plot #2. J. L. Reuscher property, Tokay variety, Lodi, California.

Treatment	Rate gal./A	Average cane weight gms. per vine	Average root-knot gall count per pint of soil
None		17.8	97.3
D-D	20[a]	32.7	39.0
D-D	40	53.1	0.4
D-D	60	36.6	10.7
EDB	6	59.4	50.3
EDB	9	60.6	24.1

[a] Figures at this dosage were obtained from vines bordering the plot in which the soil was treated by a commercial applicator before planting.

Table 3. Average cane growth and nematode counts in grape replant fumigation plot #3. D. Kirihara property, Thompson seedless variety, Livingston, California.

Treatment	Rate gal./A	Average cane weight gms. per vine	Average root-knot gall count per pint of soil
None		12.2	11.8
D-D	40	16.7	10.7
D-D	60	31.9	87.9
EDB	6	16.7	20.9
EDB	9	31.5	25.6
OS-1897	10	13.9	3.3

Table 4. Average cane growth and nematode counts in grape replant fumigation plot #4. G. Kovacevich property, Thompson Seedless variety, Madera, California.

Treatment	Rate gal./A	Average cane weight gms. per vine	Average count per pint of soil	
			Root-knot galls	Root-lesion nematodes
None		6.8	40.0	39
D-D	40	80.3	104.4	95
D-D	60	60.9	67.4	28
EDB	6	47.5	283.7	0
EDB	9	38.1	186.7	8

Table 5. Average cane growth and nematode counts in grape replant fumigation plot #5. W. B. Camp & Sons, Black Malvoisie variety, Delano, California.

Treatment	Rate gal./A	Block	Average cane weight gms. per vine	Average count per pint of soil		
				Root-knot galls		Root-lesion nematodes
				Feb. 23, 1954	May 24, 1954	
None		I	18.0	85.0	168.8	12.0
		II	18.7	0.5	0.0	9.0
		III	29.3	0.0	6.0	9.5
D-D	40	I	34.6	16.3	186.0	7.5
		II	22.0	14.7	111.0	4.5
		III	18.2	0.0	0.0	3.8
D-D	60	I	17.1	45.6	53.3	0.0
		II	20.7	0.0	0.8	10.5
		III	24.2	0.2	49.5	1.5
EDB	6	I	17.3	20.2	122.3	12.0
		II	24.7	8.6	0.0	3.0
		III	30.6	6.8	0.0	15.0
EDB	9	I	19.7	14.3	6.0	14.3
		II	32.2	18.8	92.3	18.8
		III	37.4	10.9	330.8	10.9
OS-1897	10	I	16.4	0.7	0.0	6.8
CBP-55	20	II	32.2	69.2	241.0	5.3
D-D	20	III	31.3	17.5	3.1	6.8

The lesser growth obtained after treatment with D-D at 60 gal./A may be due to injury resulting from the short interval between fumigation and planting. A longer delay may be necessary to avoid this effect of the fumigant although the vines appear to have overcome this depressed growth in the second growing season.

Plot #3: A third plot was established on the property of Mr. D. Kirihara near Livingston in a five-year-old Thompson Seedless vineyard which was very heavily infected with M. incognita var. acrita and had failed to develop to normal production in that time. The old vines were removed during January 1953, about two weeks before the fumigants were applied.

The soil type was a sandy loam with a moisture equivalent of 5.6%. The fumigants were applied February 5, 1953, when the soil moisture content was 3.8% with a temperature of 10° C at the 6-inch depth. It was not possible to obtain a roller to pack the soil after the treatments in this plot. Thompson Seedless cuttings were planted April 1, 1953.

This plot was irregular in size because of an irrigation line running diagonally across one end. Each treatment was 3 rows wide and varied between 15 and 38 vines per row.

Average gall counts from soil samples taken January 21, 1954 and average pruning weights taken the same day are presented in Table 3.

The young vines in this plot suffered to some extent during the first few months of growth in 1953 because of a hot dry sandstorm which scorched the new tender growth. Subsequent recovery of the vines seemed satisfactory.

The influence of fumigation on the growth of new vines in this plot was apparently not significant except at the higher dosages, and the plot also showed a considerable reinfestation of nematodes. The most effective nematode control was obtained from OS-1897 but it did not result in improved vines. This was probably due to phytotoxicity, which had been observed in other experiments with this chemical involving certain annual vegetable and field crops when the chemical was used at this dosage.

Recent observations near the end of the second growing season revealed a generally unsatisfactory growth and an extremely severe root-knot infection in all the treatments including the higher dosages.

Plot #4: These treatments were made in a vineyard belonging to Mr. G. Kovacevich near Madera. The area selected for the plot was in a vineyard of Thompson Seedless grapes approximately six years old and infected with both M. incognita var. acrita and Pratylenchus vulnus. The old vines were removed during January 1953.

The soil was a sandy loam with a moisture equivalent of 5.6%. The fumigants were applied February 2, 1953, when the soil moisture content was 5.6% and the temperature 10° C at the 6-inch depth. Each treatment was 3 rows wide and 20 vines long. Thompson Seedless cuttings were planted April 1, 1953.

Root-knot gall counts were obtained from soil samples collected January 21, 1954. Root-lesion nematode counts were also obtained from one-pint aliquots of this soil which were washed through 100 and 200 mesh screens. Averages of these data are tabulated in Table 4 together with the average pruning weights taken in February 1954.

The effects of fumigation on the new vines in this plot have been outstanding, almost as great as the differences due to treatment recorded in Plot #1. The coincident infestation of both root-knot and root-lesion nematodes in this vineyard may account for the extreme injury found here. The D-D treatment at 60 gal./A seems to show some degree of phytotoxicity, according to the cane growth data, but the vines appear to have overcome this depressed growth in the second growing season. Nematode control was probably less effective than in any other plot, particularly in the EDB treatments which show less growth in cane weight at the end of the first season than the D-D treatments.

Plot #5: The last experiment was established in a vineyard of Black Malvoisie grapes belonging to W. B. Camp and Sons located near Delano. These grapevines were found to be infected with M. javanica. The root-lesion nematode, P. minyus, occurred rather uniformly in the soil throughout the plot but its role in this problem has not been clearly demonstrated as yet.

The old vines were approximately 12 years old and had not been producing satisfactorily for several years. Vines in the replant plot were removed in November 1952. The soil was a sandy loam type with considerable gravel and a moisture equivalent of 8.4%. The moisture content of the soil at the time of treatment, February 17, 1953, was 5.7% with a temperature of 9.5° C at the 6-inch depth.

Three replications were included for each treatment, except D-D at 20 gal./A, OS-1897,

and CBP-55, each of which were replicated once. All treatments were 3 rows wide with 32-34 vines per row. Cuttings of Black Malvoisie grapes were planted April 2, 1953.

Root-knot gall counts were obtained from soil samples collected on February 23, 1954. Additional samples were taken by soil tube down to 3 feet on May 25, 1954, to obtain root-lesion nematode counts by soil washings and additional root-knot counts on tomatoes. The averages of these data as well as the average pruning weights taken February 23, 1954, are reported in Table 5.

There seems to be no significant influence from fumigation on the growth of these vines by the end of the first growing season, and the variation in growth throughout the plot is not correlated with the treatments. Data from this plot are reported in more detail to illustrate the uneven distribution of root-knot infestation, which indicates that the root-knot nematode is not the only cause of the general and severe decline in this vineyard. The more uniform distribution of P. minyus throughout the plot strongly suggests its implication as a cause of injury, but more information is needed regarding this species before it can definitely be considered a factor.

However, the gall counts show the root-knot nematode to be building up rapidly, and further observations on the development of the vines as well as on the populations of both nematode species will be necessary before any conclusions can be made as to the influence of nematodes in this planting or the success or failure of the chemical treatments applied.

Discussion of Results

There has been a general, and in some cases very encouraging improvement in the growth of vines planted in fumigated soil over those planted in untreated soil. However, the success of these treatments cannot be judged from the results of one year's experience but must be based on the growth and productivity of these vines over a period of years.

The most promising results were found in Plot #1 where removal of the old vineyard was followed by a rotation of 3 years before the soil was fumigated. Many growers believe that vines grown in nematode-free soil for 2 to 3 years can become well-enough established to withstand subsequent nematode infections and still remain vigorous and productive. Even if this should be true, it must still be determined whether the advantage of more effective control obtained in Plot #1 will be maintained or whether the nematodes will build up to high population levels in time to cause injury. Also, this advantage of rotation in obtaining superior vine growth as well as nematode control must be confirmed by additional experience in other areas and under varying conditions, including different soil types, grape varieties, nematode species, etc.

The nematode control obtained in Plots #2, #3, #4 and #5 is not consistent and should probably be considered as generally unsatisfactory. This failure of control is correlated with the removal of the old vines within 3 months or less previous to the time of treatment, and there is a good probability that survival of the nematodes occurs within the roots of the old vines that may persist in the soil for months or even years. This suggests that the chemicals at the dosages used are not able to penetrate the root-tissues to effect the desired mortality.

The nematode counts show definitely that in most cases nematode infections of the new vines occurred during the first year of growth. There is serious doubt, therefore, as to the eventual success of these fumigation treatments, even though the vines in some of the treatments are very vigorous and have continued to make satisfactory progress during the second year of growth.

Although most growers prefer to replace old vineyards immediately following their removal it is likely that a rotation program followed by soil fumigation will be essential in order to maintain productive vineyards for the length of time necessary to make them profitable. The minimum time for such rotations has not been determined but in this case 3 years seems to be sufficient.

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CONTROL OF ENDOPARASITIC ROOT NEMATODES IN STRAWBERRY PROPAGATION STOCKS BY HOT-WATER TREATMENTS

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Abstract

To control the northern root-knot nematode, Meloidogyne hapla, and a meadow nematode Pratylenchus penetrans, which are widely distributed on strawberry propagating stocks in the United States, hot-water treatments were investigated. The thermal death times of M. hapla, determined from root samples of plants grown for 60 days after hot-water treatment, were longer than those determined from root samples taken immediately after treatment. These in turn were longer than periods reported for detached egg masses and free-swimming larvae of one species of root-knot nematode by previous investigators. The thermal death time curve of M. hapla determined from plants 60 days after treatment is approximately a straight line when temperature is plotted against time on a logarithmic scale between the points 121° F. for 420 seconds and 140° F. for 6 seconds. The thermal death points of P. penetrans and M. hapla were almost identical as determined from root samples taken immediately after treatment. At several different hot-water treatments sufficient to kill the endoparasitic root nematodes, dormant Blakemore strawberry plants survived without injury. Most Blakemore plants that were not dormant at the time of treatment were killed by any treatment sufficient to kill these nematodes. When dormant Blakemore plants were treated in hot water at 127° F. for 120 seconds on March 3, surface-dried, and returned to cold storage before they were planted on April 2 most mother plants survived and the number of runner plants produced during the summer was not significantly different from untreated control plants. However, when dormant plants were subjected to the same treatment on April 2 and planted immediately, some mother plants died and the number of runner plants produced during the summer was significantly less than the untreated control plants.

Since 1909, when Marcinowski (20) found that hot-water treatments sufficient to kill the foliar nematode, Aphelenchoides olesistus (Ritzema Bos) Steiner, were tolerated by ferns and begonias, considerable work has been reported on hot-water treatments for control of various nematodes. Control of the bud and leaf nematodes, Aphelenchoides spp., of strawberry runner plants by hot-water treatment is standard practice in Europe (9, 25) and control of bulb and stem nematodes, Ditylenchus spp., by hot-water treatments in the production of bulbs is widely used (11).

Hot-water treatments for the control of endoparasitic root nematodes have been reported several times, but standarized procedures have apparently not been published. Byars (4) first reported control of a root-knot nematode by hot-water treatment of dasheen tubers in 1917. In 1929 Cobb (7) reported the control of root-knot nematodes by hot-water treatments of the roots of plants of 16 different orders. Unpublished records of the U. S. Department of Agriculture, Horticultural Crops Research Branch, Nematology Section, show that one of these was the cultivated strawberry. Hot-water treatment of strawberries for root-knot nematodes did not become a standard practice, apparently because strawberry plants did not always withstand the suggested treatment of 118° F. for 20 minutes. Hot-water treatments have been tried with varying degrees of success for the control of root-knot nematodes on black locust (6), gladiolus (28), iris (28), lavender (18), mulberry (29), peony (21), rooted rose cuttings (22), sweetpotato (2), tobacco (28), tuberose (24, 27, 28), and tung (28). Hot-water treatment for the control of meadow nematodes in lily-of-the-valley rhizomes has also been reported (11, 12).

Both the northern root-knot nematode, Meloidogyne hapla Chitwood, and the meadow nematode, Pratylenchus penetrans (Cobb) Sher & Allen, are widely distributed on strawberry planting stocks. The northern root-knot nematode reduces root growth and probably brings about considerable damage to strawberry plants, especially during periods of summer drought. Campbell (5) reported that strawberry plants are devitalized or even killed by heavy infections of this root-knot nematode. Horn and Martin (16) noted that this root-knot nematode caused

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severe damage to strawberry roots and that both growth and yield were increased when it was controlled. The meadow nematode is probably even more serious than the northern root-knot nematode on strawberries. Hildebrand (13) and Hildebrand and West (14) found meadow nematodes associated with strawberry root rot in certain soils in Canada. Klinkenberg (19) suggested that meadow nematodes are possibly the primary cause of strawberry black-root disease in the Netherlands. It appears desirable that control measures for both these endoparasitic root nematodes be developed to provide vigorous planting stocks and to prevent contamination of clean fields.

Hoshino and Godfrey (17) determined the thermal death time curve of free-swimming larvae and of detached egg masses of one of the root-knot nematodes. Hastings (12) determined the minimum lethal temperature for meadow nematodes in oat seedling roots for a 10-minute immersion.

The thermal death time points of dormant strawberry plants have apparently not been studied in detail. Hodson (15) reported complete killing of actively growing plants immersed in water at 118° F. for 5 minutes. Danish investigators (9) found that non-dormant strawberry plants would withstand hot-water treatments of 113° to 116.6° for 14 minutes with only slight damage while similar plants treated at 118.4 to 122.2° for 10 minutes were severely damaged or killed.

This paper presents data on the thermal death time of the northern root-knot nematode (M. hapla) in hot-water-treated strawberry plants and of the meadow nematode (P. penetrans) in strawberry roots sampled immediately after treatment, as well as on the hot-water tolerance of Blakemore strawberry plants.

MATERIALS

Strawberry plants of the Blakemore variety were used in the root-knot nematode and dormant-plant hot-water tolerance studies. These plants were severely infected with northern root-knot nematodes and lightly infected with meadow nematodes. The plants were dug in February, bundled and held in cold storage at 30° F. until used for heat treatments. Other plants of this variety, dug in April just prior to treatment, were used in the hot-water tolerance studies of non-dormant plants. Plants of the Robinson variety, infected with meadow nematodes only, were used in the meadow nematode studies. The plants were dug in April after spring growth had started.

The hot-water bath used was a thermally insulated, metal tank of about 8-gallon capacity equipped with electric heating coils. Temperature was controlled to ± 0.5° F. A stirrer provided circulation to maintain an even temperature throughout the bath. Plants were placed loosely in wire baskets and completely immersed in the tank for specific periods at the different temperatures. Plants were immediately plunged into cold running tap water after treatment.

METHODS

Root-knot Nematode Studies

Two experiments were used to locate the thermal death points of the root-knot nematode at representative temperatures between 112° and 150° F. In the first experiment, on March 3, 1954, 26 infected plants were treated by each of the following temperature-time combinations: 112°--900, 1800, and 3600 seconds; 115°--600, 900, 1200, and 1800 seconds; 118°--300, 600, and 900 seconds; 121°--60, 120, 300, and 600 seconds; 124°--60, 120, and 300 seconds; and 127°--60 and 120 seconds. One plant was taken for root sampling and the other 25 were planted immediately following treatment, 5 per 6-inch pot, in steam-sterilized soil and then grown on a slat bench in the greenhouse.

The survival of nematodes immediately following treatment was determined by placing a 1-gm. sample of roots from the single plant in a petri dish and covering the roots with distilled water. The dishes were examined periodically under a microscope for approximately 1 month. When root-knot nematodes were not killed, larvae or adult males migrated from the roots and settled in the bottom of the dish. When they were apparently killed, no living forms could be found on the bottom of the dish during the period of observation.

The survival of nematodes was checked after the treated plants had grown for 60, 150, and 180 days. Sixty days equaled at least one generation of the root-knot nematode (10). At each of these periods, a single pot containing 5 plants was selected from each treatment at random

and freed of soil in running tap water. The roots were examined immediately for galls and egg masses of the root-knot nematode and petri dish samples were taken and observed over a period of one month to check for living forms.

In the second experiment, on March 24, 1954, 11 infected plants were treated by each of the following temperature-time combinations: 118°--600, 750, and 900 seconds; 121°--150, 210, 300, and 420 seconds; 124°--90, 120, 150, and 210 seconds; 127°--30, 42, 60, 90, 120, and 180 seconds; 130°--15, 30, 60, and 120 seconds; 135°--1, 6, 30, and 120 seconds; 140°--1, 6, and 90 seconds; 145°--1, 6, and 60 seconds; and 150°--1, 6, and 60 seconds. In addition, a single plant was treated by the following combinations: 121°--255 seconds; 124°--75, 105, and 135 seconds; 127°--36, and 50 seconds; 130°--10, 21, and 42 seconds; 135°--2.4, 15, and 60 seconds; 140°--2.4, 15, and 38 seconds; 145°--2.4, 15, and 30 seconds; and 150°--2.4, 15, and 30 seconds. Root samples for determination of nematode survival immediately after treatment were taken from the single plants and from 1 plant of the 11. The other 10 plants were planted in steam sterilized soil, 5 per 6-inch pot, and grown for 60 days. The potted plants were then examined for living root-knot nematodes as in the previous experiment.

Meadow Nematode Studies

Thermal death time points of the meadow nematode in Robinson strawberry roots were determined at 3° intervals from 115° to 130° F. Prior to treatments, a few roots from each plant were sampled in order to be certain that meadow nematodes were present. Single heavily infected plants were selected and subjected to the following temperature-time treatments: 115°--2700, 3600, and 5400 seconds; 118°--900, 1200, 1500, and 1800 seconds; 121°--300, 360, 450, and 600 seconds; 124°--105, 120, 150, and 210 seconds; 127°--36, 45, 54, 60, 75, and 90 seconds; and 130°--15, 22.5, 30, 42, and 60 seconds. Following treatment, 1-gm. root samples were placed in petri dishes and examined periodically for the emergence of migrating nematodes for approximately 1 month. As Hastings (12) previously reported, meadow nematodes did not migrate from some heat-treated roots for 2 or 3 weeks.

Hot-water Tolerance of Strawberry Plants

The effect of hot-water treatments on dormant Blakemore strawberry plants was evaluated in the plants that were grown following treatments for root-knot nematodes. Survival was noted and the plants were given an arbitrary rating at the time of harvest based on new growth and general appearance. Ratings were given on the following basis: Dead = 0, control = 10. If treated plants appeared better than the controls, a relative rating higher than 10 was given.

In another experiment the heat tolerance of dormant Blakemore strawberry plants was further checked by more severe treatments. Five cold-storage plants were treated on April 6, 1954, by each of the following temperature-time combinations: 118°--1500, 2100, 3000, and 4200 seconds; 121°--750, 1050, 1350, and 1800 seconds; 124°--450, 750, 900, and 1200 seconds; and 127°--240, 300, 420, 600, and 900 seconds. The 5 plants from each treatment were planted in a 6-inch pot and grown in the greenhouse until June 18, at which time the surviving plants were counted and rated.

The effect of heat treatment on the survival and production of runner plants in the field was checked in another experiment. In this experiment the effect of the dormancy of the plants at the time of treatment on the runner plant production was also checked. On March 3, 1954, cold-storage Blakemore plants were treated by the following temperature-time combinations: 118°--600 seconds; 124°--300 seconds; and 127°--60 and 120 seconds. Following treatment, the plants were surface-dried, placed in polyethylene bags and returned to cold storage. On April 2, other cold-storage plants and freshly dug plants were treated by the same temperature-time combinations. These 3 lots of plants were set in the field the same day in 4 replications of 4 plants each in split plots with whole-plot comparison between conditions of dormancy at time of treatment and sub-plot comparison between temperature-time treatments. On June 8 plant-survival data were taken and on September 21 runner-plants were counted.

RESULTS

Thermal Death Time of Root-knot Nematodes in Strawberry Root Samples

The maximum survival periods and the minimum lethal periods of the root-knot nematode in strawberry roots at several temperatures as determined immediately after hot-water treat-

Table 1. Comparison of Hosino and Godfrey (1933) data on hot-water treatments of root-knot nematode larvae and egg masses with results obtained from hot-water treatments for control of the northern root-knot nematode in strawberry roots.

Treatment temperature	Data of Hoshino and Godfrey				Nematodes in roots of strawberry plants, samples taken--			
	Free-swimming larvae		Detached egg masses		Immediately after treatment		From plants grown 60 days or more after treatment	
	Maximum survival period	Maximum lethal period	Maximum survival period	Maximum lethal period	Maximum survival period	Maximum lethal period	Maximum survival period	Maximum lethal period
°F.	Seconds	Seconds	Seconds	Seconds	Seconds	Seconds	Seconds	Seconds
112	318	333	1863	2055	3600		3600	
115	218	233	811	871	1200	1800	1800	
118	79	86	413	473	600	900	900	
121	53	58	214	274	300	420	300	420
124	6	7	57	114	105	120	150	210
127	0	1	30	48	42	50	60	120
130			1	5	15	21	30	60
135			2	3	2.4	6	6	30
140					1	2.4	1	6
145					1	2.4	1	6
150					1	2.4	1	6

ments and after the treated plants had been growing for 60 or more days are given in Table 1. These periods are greater than those for detached root-knot nematode egg masses calculated from the data of Hoshino and Godfrey (17). The periods of survival and death of the nematodes in the plants grown for at least 60 days following hot-water treatments are greater than those indicated by root samples taken immediately after treatment. The increase in these periods may indicate that a certain degree of protection from heat is afforded by the tissues of the strawberry roots. It might also indicate that the small root samples used were not sufficient to detect nematodes when the population was small. From the data given for the maximum survival period and the minimum lethal period of the nematodes in plant roots grown for 60 days or more after treatment, the approximate thermal death time curve of the root-knot nematode in strawberry plants can be constructed. On a semi-log graph this curve approaches a straight line between 121° and 140° F. (Fig. 1).

Thermal Death Time of Meadow Nematodes in Strawberry Root Samples

The maximum survival periods and the minimum lethal periods of meadow nematodes in strawberry roots at several temperatures as determined by root samples taken immediately after heat treatments are given in Table 2. Between 121° and 130° these periods are very nearly the same as the corresponding ones of the northern root-knot nematode determined by root samples taken immediately after treatment. The thermal death time curve of the meadow nematode is probably very nearly the same as that of this root-knot nematode.

Hot-water Tolerance of Strawberry Plants

The survival of dormant Blakemore strawberry plants and the subsequent ratings of the surviving plants 60 days or more after various hot-water treatments sufficient to control endoparasitic root nematodes are given in Table 3. Many plants in which heat injury was initially observed recovered and after a short time appeared better than the nematode-infected control plants. It is apparent that several hot-water treatments could be used to control endoparasitic root nematodes in strawberry plants.

Certain temperature-time treatments sufficient to control endoparasitic root nematodes

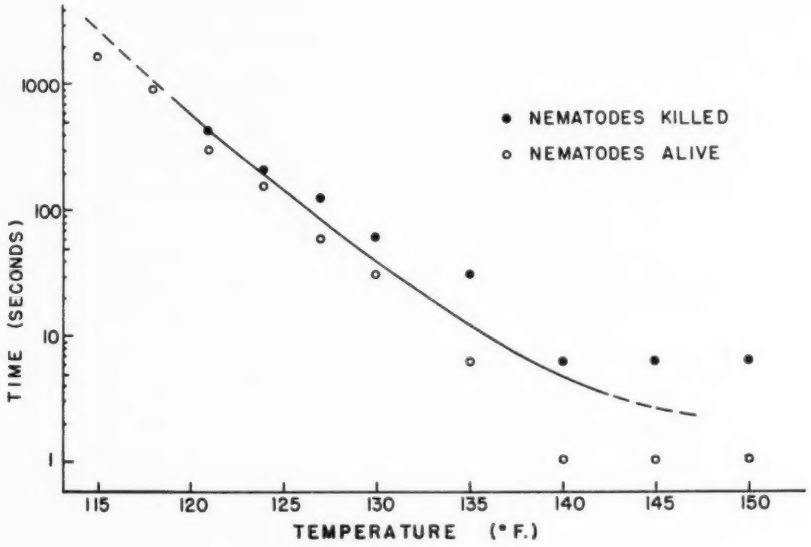

FIGURE 1. The thermal death time curve of Meloidogyne hapla, the northern root-knot nematode, determined from root samples of strawberry plants grown for 60 days after hot-water treatments.

Table 2. Maximum survival and minimum lethal periods of the meadow nematode after hot-water treatment, as indicated by strawberry-root samples taken immediately after treatment.

Treatment temperature	Maximum survival period	Minimum lethal period
° F.	Seconds	Seconds
115		2700
118	900	1200
121	360	450
124	120	150
127	45	60
130	15	22.5

when applied to dormant plants do not affect plant survival or runner-plant production in the field, but when applied to non-dormant plants the same treatments kill all plants. Table 4 gives the results of plant survival and runner-plant production following 4 different temperature-time treatments of plants in 3 conditions of dormancy. In this study treatment at 118° for 600 seconds was selected because it was sub-lethal to root nematodes; 127° for 60 seconds was approximately along the root nematode-control curve; 127° for 120 seconds was adequate for controlling root nematodes without serious damage to plants grown in the greenhouse; and 124° for 300 seconds was slightly injurious to plants grown in the greenhouse. At 127° for 120 seconds and 124° for 300 seconds, plant survival and runner-plant production were significantly

Table 3. Survival of dormant Blakemore strawberry plants after hot-water treatments sufficient to control endoparasitic root nematodes in plants and the ratings of plants after 60 days or more.

Treatment		Plants treated	Plants survival	Rating[a] of the surviving plants
Treatment	Period			
°F.	Seconds	Number	Number	
121	420	10	10	12
	600	25	10	8
	750	5	5	13
	1050	5	5	12
	1800	5	0	0
124	210	10	10	10
	300	25	19	7
	450	5	5	10
	1200	5	0	0
127	120	35	35	12
	180	10	9	11
	240	5	5	8
	300	5	5	7
	600	5	0	0
130	30	10	10	11
	60	10	10	11
135	30	10	9	9
	120	10	0	0
140	6	10	10	9
	90	10	1	1

[a] Arbitrary rating of growth and appearance made at the time of harvest on the following basis: Dead = O, untreated control = 10. If treated plants appeared better than the controls, a relative rating higher than 10 was given.

reduced when the plants were treated just prior to planting. However, when the plants were treated early, surface-dried, and returned to cold storage before planting little or no injury resulted from 127° for 120 seconds. Earlier treatment was significantly better than later treatment at 127° for 120 seconds and highly significantly better at 124° for 300 seconds. Least damage resulted when strawberry plants were completely dormant at the time of hot-water treatment.

DISCUSSION

Soil fumigation to kill most plant parasitic nematodes in infested fields before planting is possible. Allen and Raski (1) reported that a split application of DD at the rate of 80 gallons per acre completely controlled meadow nematodes in a strawberry field for at least 4 months. For several years some nurserymen have made a practice of fumigating the fields in which they propagate their strawberries each year, but plants from these fields are often heavily infected with endoparasitic root nematodes when removed. Reinoculation of these fields appears to result in major part from the use of nematode-infected mother plants. The quantity production of substantially nematode-free strawberry plants requires nematode-free planting stocks.

Three ways of controlling endoparasitic root nematodes in strawberry mother plant stocks have been tried. (a) Darrow and Waldo (8) reported that runner plants rooted in heavy soils

Table 4. Survival and runner-plant production of Blakemore strawberry plants as influenced by hot-water treatments applied to non-dormant plants and to dormant plants on two different dates.

Plants used	Treatment		Mean survival for plots of 4 plants	Mean runner plants rooted Sept. 21
	Temperature	Period		
	°F	Seconds	Number	Number
Winter dug, cold stored, treated March 3, 1954, surface-dried and returned to cold storage, planted April 2, 1954.	-	a	4.00	98.0
	118	600	4.00	84.5
	124	300	3.25	63.5
	127	60	3.75	81.5
	127	120	3.75	80.0
Winter-dug, cold-stored, treated and planted April 2, 1954.	-	a	4.00	77.2
	118	600	4.00	89.2
	124	300	0.25	2.2
	127	60	3.75	79.5
	127	120	3.00	48.2
Dug, treated, and planted April 2, 1954.	-	a	4.00	92.2
	118	600	0.00	0.0
	124	300	0.00	0.0
	127	60	0.00	0.0
	127	120	0.00	0.0
L.S.D. 5%			0.66	25.5
L.S.D. 1%			0.88	34.2

a Untreated control plants.

were free of root-knot nematodes even when mother plants were heavily infected because root-knot nematode larvae move slowly through such soils. (b) In experimental work at Beltsville, runner plants were rooted in steam-sterilized soil in the greenhouse. These were then increased in a field of fumigated soil. (c) Reimer and Davidson (23) set infected mother plants in large tin cans placed in a field of methyl-bromide-fumigated soil. As soon as runner plants were established, the cans were removed and the plants subsequently produced were substantially free of nematodes. In each case however, a reservoir of live nematodes was in close proximity to the runner plants so that there was danger that new stocks might become contaminated. Though the first way has been used at times, it has apparently not proved very practical. A few of the plants dug from the Beltsville field (second way) were found to be heavily infected with root-knot nematodes. An admittedly serious drawback of the third way is that heavy rains before the cans are removed may cause contamination of the runner plants.

The experiments reported in this paper indicate that control of endoparasitic root nematodes in propagating stocks of the Blakemore strawberry variety is possible and practical by hot-water treatments. From preliminary observations, it appears that other strawberry varieties will show heat tolerance similar to that of the Blakemore variety.

In Table 3 are given several temperature-time treatments which controlled both the meadow and the northern root-knot nematode in dormant propagation stocks without seriously injuring them. If plants are completely dormant, certain treatments, such as 127° for 120 seconds will not weaken them. Plants given such treatment grew as well in the field and produced runner plants in as great abundance as untreated plants. If the soil is fumigated plants thus produced will have the considerable advantage that they will be substantially free of endoparasitic root nematodes.

For practical control of the endoparasitic root nematodes, temperature treatments below 130° F. should probably be used. The period necessary to kill the nematodes at 140° is apparently very nearly the same as the period at 150°. This indicates that some time is necessary for heat to penetrate the root tissues to affect the nematodes. Staniland (25) reported that crowns of large strawberry runner plants are heated to the temperature of a water bath at 115° F. in 3 to 4 minutes, while smaller ones are heated through in 2 to 3 minutes. Brief

treatments, such as 140° F. for 6 seconds, were apparently effective in these experiments because root-knot and meadow nematodes parasitize mainly the cortical tissues of roots (3, 26).

Dormant strawberry plants infected with cyclamen mites, Tarsonemus pallidus, or with other nematodes (Aphelenchoides fragariae and Ditylenchus dipsaci) were not available for experimentation. Since the thermal death time points of these (9, 15, 25) are apparently below those of the endoparasitic root nematodes, treatment for the root nematodes will probably be effective for these other strawberry pests, provided temperature-time combinations that permit sufficient heat penetration to reach pests are selected.

Quantity production of nematode-free planting stock is the distinct advantage of hot-water therapy. Large acreages can be set with nematode-free plants, and if the soil has been fumigated and care is taken not to reinfest it by various cultural practices, the entire stock of plants produced at the end of a season should be substantially free of nematodes. Annual hot-water treatment of propagation stock would control chance recontamination and subsequent nematode build-up.

The successful hot-water treatment of dormant strawberry plants can possibly be adapted to other clonally propagated plants plagued with endoparasitic root nematodes. Comparisons of thermal death times of the nematodes in the roots of treated plants with the thermal injury of dormant plants can be made to determine whether hot-water therapy is feasible.

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INACTIVATION OF NON-PERSISTENT VIRUSES IN STRAWBERRY PLANTS BY HOT-AIR TREATMENT[1]

P. W. Miller[2]

Abstract

In cooperative experiments conducted by the U. S. Department of Agriculture and the Oregon Agricultural Experiment Station at Corvallis, Oregon, certain non-persistent strawberry viruses (crinkle or yellows virus complexes or both) were inactivated in infected plants by hot-air treatment. Inactivation occurred at temperatures of 34° to 38°, both ±1°, for varying periods. After hot-air treatment at 33°-35° for 114 to 192 hours, there were 11 plants free of non-persistent viruses; and for 193 to 342 hours, 2. At 34°-36° for 43 to 96 hours, there was 1; for 104 to 168 hours, 3; and for 238 to 408 hours, 2. At 35°-37° for 66 to 96 hours, there were 2; 99 to 191 hours, 10 , and for 198 to 432 hours, 8. At 36°-38° for 31 to 95 hours there were 18; for 97 to 191 hours, 57; for 194 to 288 hours, 26; and for 312 to 432 hours, 7. At 37°-39° for 69 to 103 hours there were 6; for 113 to 174 hours, there were 4, and for 190 to 266 hours, 6 plants free of non-persistent viruses. A temperature-time period that gave a relatively high percentage of plants free of non-persistent viruses (87 percent) and plant survival (65 percent) was 36°-38° for 194 to 288 hours. A total of 482 plants was heat-treated; 206 of the plants, or 43 percent, survived the heat treatment and were tested for the presence of non-persistent viruses; 166 of these plants, or 81 percent, were found free of non-persistent viruses -- a very high percentage of inactivation.

The Brightmore, Sierra, Cupertino, Utah, and York varieties were found to be more heat-sensitive than the Marshall, Northwest, Narcissa, Redheart, Tahoe, and Oreg.-USDA selections 2172 and 2242.

Well-established plants were found by test to withstand higher temperatures for longer periods than newly-planted ones.

By means of hot-air treatments, non-persistent viruses were inactivated in some plants of the following strawberry varieties: Marshall, Northwest, Redheart, Tahoe, Narcissa, Utah, Sierra, Cupertino, Brightmore, York, and Oreg.-USDA selections 2172 and 2242.

The thermal inactivation of viruses in plants has been investigated by a number of workers. Literature on this subject has been recently reviewed by Miller (5).

R. H. Fulton (3) was the first investigator to report on attempts to inactivate viruses in infected strawberry plants by hot-water treatment; he was unable to inactivate type 2 virus of Demaree and Marcus (1) in the Robinson strawberry by immersing infected plants in hot water at 45° C. for 30 minutes. In 1953, however, Miller (5) obtained limited inactivation of viruses in the Marshall and other strawberry varieties by hot-water treatment at 43° to 48° for immersion periods of 1/2 to 7 hours, but the percentage of plants surviving the treatment was relatively low. In 1953, Posnette et al. (6) demonstrated the inactivation of strawberry virus 1 (mild crinkle), virus 3 (crinkle), and an undescribed virus causing a severe leaf necrosis of the Royal Sovereign strawberry by holding infected plants in a hot-air chamber at 37° for 8 days. In 1954, J. P. Fulton (2) reported the inactivation of type 1 and type 2 viruses of Demaree and Marcus in the Blakemore strawberry by hot-air treatment at 38° to 42° for 3 months after an acclimation period at 27° for several weeks. In the same year, R. H. Fulton (4) also demonstrated the inactivation of type 2 virus of Demaree and Marcus in infected Robinson strawberry plants by hot-air treatment at 36° to 40°. The lowest temperature for inactivation was 36° for 8 days; the most effective temperature-time period was 38° for 8 days.

Experiments on the inactivation of strawberry viruses by hot-air treatment were begun at Oregon State College in 1953 and have continued sporadically since then. A report on investigations carried on and the results obtained to date follows.

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[1] Approved for publication by the Director of the Oregon Agricultural Experiment Station and by the Chief of the Horticultural Crops Research Branch, Agricultural Research Service, U. S. Department of Agriculture. Contribution of the Department of Botany and Plant Pathology of the Oregon Agricultural Experiment Station and Horticultural Crops Research Branch, Agricultural Research Service, U. S. Department of Agriculture.

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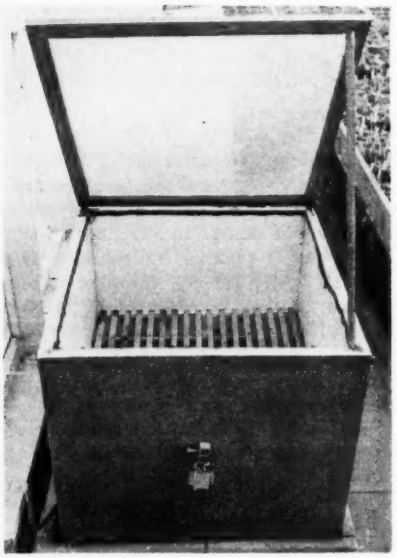

FIGURE 1.
The thermostatically controlled hot-air chamber used in strawberry virus inactivation investigations.

Methods

A chamber measuring 3 x 3 x 3-1/2 feet was constructed of plywood with double walls separated by rockwool for insulation. The top was hinged to permit access to the inside of the chamber and fitted with a glass plate to allow light to enter (Fig. 1). The air in the chamber was heated by a wire heating coil fastened to the bottom of a slatted platform raised about 6 inches above the bottom. The air temperature was maintained at ±1°C. by a thermo-regulator. The capacity of the chamber was 30 plants in 4-inch pots.

Well-established, potted strawberry plants definitely known to be infected with strawberry virus complexes (yellows or crinkle complexes or both) were placed in this chamber maintained at various temperatures for varying periods (Table 1). Approximately 30 percent of the plants treated were infected Marshall; the remainder were infected Northwest, Brightmore, Redheart, Sierra, Utah, Tahoe, Cupertino, Narcissa, and York varieties and Oreg.-USDA hybrid selections 2172 and 2242. All plants treated had previously been shown to be viruliferous by grafting to Fragaria vesca indicator plants or by transferring viruses to F. vesca by aphids.

The temperatures used varied from 34° C. to 38°, both ±1°, and the time of exposure from 31 to 432 hours.

One to three weeks after heat treatment, the surviving plants were tested for the presence of viruses by caging virus-free aphids on them and 6 to 10 days later tranferring them to healthy F. vesca indicator plants where they were permitted to feed for 4 to 6 days longer. The plants which proved symptomless after the first indexing were retested by the use of aphids two additional times. A small percentage (less than 5 percent) of the treated plants produced runners; these were further tested by grafting to F. vesca. A few of the plants (less than 2 percent) that did not show any evidence of virus infection on the first indexing showed symptoms on the second indexing. However, none of the plants apparently free of non-persistent viruses after the second indexing showed virus symptoms thereafter. If no virus symptoms appeared on any of the indicator plants after three indexings it was presumed that the plants were free of all non-persistent viruses. However, they may not be free of all persistent virus components as Prentice and Woollcombe (7) recently showed that from the time of the first feeding on plants infected with strawberry virus 3, 10 to 19 days elapsed before the aphids became infective -- a longer latent period than any previously recorded for any other component.

Results

The results of the hot-air treatments are summarized in Table 1. In Table 2 are given the varietal differences in heat tolerance.

Table 1. Surviving and virus-free[a] strawberry plants after treatment for various periods with hot air (33° to 39° C.) Corvallis, Oregon, 1953-54.

Approximate temperature range (°C)[b]	Treatment period range	Plants treated	Plants surviving	Surviving plants free of non-persistent viruses[c]
	Hours	Number	Number	Percent
33-35	48-95	7	1	0
	114-192	29	13	84.6
	193-342	3	2	100
34-36	43-96	18	3	33.3
	104-168	16	4	75.0
	238-408	6	2	100
35-37	66-96	15	3	66.6
	99-191	42	18	55.5
	198-432	13	12	75.0
36-38	31-95	68	25	72.0
	97-191	170	70	81.4
	194-288	46	30	86.6
	312-432	9	7	100
37-39	69-103	10	6	100
	113-174	19	4	100
	190-256	11	6	100
Totals		482	206	81.3

[a] Free of non-persistent viruses.

[b] Temperature set for midpoint of range and controlled ± 1° C.

[c] Freedom from non-persistent viruses determined by aphid transfer 1 to 3 weeks after hot-air treatment and 2 subsequent reindexings. (Vector acquistion feed period on host 6 to 10 days; post-acquisition feed period on Fragaria vesca 5 to 10 days). A small percentage (less than 5 percent) of the treated plants were also tested by grafting to F. vesca. Further details in text.

After hot-air treatment at 33° to 35° C. for 48 to 95 hours there were no plants free of non-persistent viruses; for 114 to 192 hours, there were 11; and for 193 to 342 hours, 2. At 34° to 36° for 43 to 96 hours, there was 1; for 104 to 168 hours, 3; and for 238 to 408 hours, 2. At 35° to 37° for 66 to 96 hours, there were 2; 99 to 191 hours, 10; and for 198 to 432 hours, 8. At 36° to 38° for 31 to 95 hours there were 18; for 97 to 191 hours, 57; for 194 to 288 hours, 26; and for 312 to 432 hours, 7. At 37° to 39° for 69 to 103 hours, there were 6; for 113 to 174 hours, there were 4, and for 190 to 266 hours, 6 plants free of non-persistent viruses. A temperature-time period that gave a relatively high percentage of plants free of non-persistent viruses (87 percent) and plant survival (65 percent) was 36° to 38° for 194 to 288 hours.

There are a number of inconsistencies in both the plant-survival and the virus-inactivation data. The inconsistencies in plant survival may be due to varietal differences or to differences in the physiological condition of the various plants. The inconsistencies in virus inactivation may be associated with the presence in the different plants of different virus components or mixtures thereof which have different thermal death points. Posnette et al. (6) reported that different strawberry viruses have different thermal death points.

A total of 482 plants was heat-treated; 206 of the plants, or 43 percent, survived the heat treatment and were indexed for viruses; 166 of these plants, or 81 percent, were found to be free of non-persistent viruses. This is a very high percentage of inactivation and points to the efficiency of the method as a means of obtaining virus-free mother plants for foundation stocks,

Table 2. Surviving and virus-free[a] plants of various strawberry varieties treated with hot air (33° to 39° C.) for various periods, Corvallis, Oregon, 1953-1954.

Variety or selection number	Plants treated	Plants surviving	Surviving plants free of non-persistent viruses[b]
	Number	Percent	Percent
Oreg.-USDA 2172	14	71.4	100
Oreg.-USDA 2242	5	60.0	33.3
Marshall	166	57.2	73.5
Tahoe	7	57.1	75.0
Redheart	54	53.5	75.8
Northwest	102	47.0	93.7
Narcissa	60	41.6	72.0
Utah	12	33.3	100
Sierra	7	28.6	100
Brightmore	51	27.4	80.0
York	7	14.3	100
Cupertino	11	9.1	100

[a] Free of non-persistent viruses.

[b] Freedom from non-persistent viruses determined by aphid transfer 1 to 3 weeks after hot-air treatment and 2 subsequent reindexings. (Vector acquisition feed period on host 6 to 10 days; post-acquisition feed period on Fragaria vesca 4 to 6 days.) A small percentage (less than 5 percent) of the treated plants were also tested by grafting to F. vesca.

at least in those cases where all plants of certain varieties are now infected with non-persistent viruses.

Of the 166 plants found free of non-persistent viruses, 104 subsequently died from the ill-effects of the heat treatment leaving 62 plants still alive; all of these plants are apparently still free of non-persistent viruses 4 to 8 months after treatment.

Some varieties were found to be more heat-susceptible than others (Table 2). Thus, the Brightmore, Sierra, Cupertino, Utah, and York varieties were found to be more heat-sensitive than the Marshall, Northwest, Redheart, Narcissa, Tahoe, and Oreg.-USDA selections 2172 and 2242.

As Posnette et al. (6) first reported, well-established plants were found by test to withstand higher temperatures for longer periods than newly-planted ones.

By means of hot-air treatments, non-persistent viruses have been inactivated in some plants of the following strawberry varieties: Marshall, Northwest, Redheart, Tahoe, Narcissa, Utah, Sierra, Cupertino, Brightmore, York, and Oreg.-USDA selections 2172 and 2242.

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RASP LEAF VIRUS DISCOVERED IN NEW MEXICO ON SWEET CHERRY

Paul C. Duffield and Philip J. Leyendecker

Raspleaf, a virus disease of sweet cherry (Prunus avium L.), has been unquestionably identified in southern New Mexico. The infected trees (Lambert variety) were found in a 20-year-old orchard at Mountain Park, New Mexico, located on the western slope of the Sacramento Mountains. Eight localized trees in a 200-tree orchard were found to be infected. A survey of other cherry orchards in the vicinity did not reveal the presence of additional infected trees.

This disease was first observed on sweet cherry in Colorado in 1935 but its virus nature was not determined until 1942 (1). Rasp leaf has since been reported from Washington, Montana, Idaho, Utah, and California, occurring on the Lambert, Napoleon (Royal Ann), Bing, Black Tartarian, Mazzard, and Montmorency varieties.

FIGURE 1. Appearance of extreme distortion, folding, and rasp-like symptoms of virus infected sweet cherry leaves.

The most distinguishing symptom of the disease is the abnormal rasp-like outgrowths between the veins on the under surface of the leaves. Severely affected leaves are small, narrow, and distorted, and usually fold together on the upper surface. The folded leaves give the trees an appearance of having been defoliated. Reduction in terminal growth is also a common symptom, as evidenced by a rosetting of leaves on the lateral branches of infected trees. These symptoms can be seen in Figures 1 and 2. The fruit is normal in size and texture but production is much reduced in comparison with healthy trees.

Among the eight diseased trees there was a great difference in the extent of infection. Two trees were infected throughout while the other six trees showed from two to three branches to as much as two-thirds of the crown infected. This suggested that the virus could have been spread from tree to tree by an insect vector. The virus appeared to move from the center of the trees outward into the branches. Movement would seem to have been rather rapid since the orchardist reported that he had first seen the disease only about 4 or 5 years ago. The time and source of the infection is in question as the trees were budded locally 19 or 20 years ago, and Bodine (2) reported the incubation period of rasp leaf to range from 9 months to 2 years.

Upon learning the true nature of the disease the orchardist removed and destroyed the infected trees. The remaining trees will be kept under observation for any further development of the disease.

FIGURE 2. Rasp-leaf infected sweet cherry tree showing the defoliation-like symptoms and the progression of the virus from the center of the tree towards the branch terminals. Note rosette-like effect resulting from reduced terminal growth.

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THE PHONY PEACH VIRUS IN MISSOURI

D. F. Millikan[1] and Julius R. Anderson[2]

In an effort to control the phony peach virus disease in the peach-growing area of southeastern Missouri, annual Federal and State surveys have been made since 1930. However, it was not until 1932[3], when 20 diseased trees were found in New Madrid and Pemiscot Counties, that the disease was actually identified. The greatest number of diseased trees was found in 1936 when 219 infected trees were located in 14 counties. Sixty-eight diseased trees were reported in 1937, but since that time incidence has always been less than 30 trees in any one year. By 1944 all counties except Pemiscot and Dunklin had been released from quarantine on the basis that no disease had been found in them for 3 years. In 1946, Pemiscot was declared free from the disease, leaving only Dunklin County under quarantine. Emphasis since 1949 has shifted from the southern part of Dunklin County where peaches are grown in home orchards to the commercial plantings around Campbell.

Interest in peach production has declined since 1949 when 16 orchards with 7,438 trees were removed. Recently, interest has revived somewhat inasmuch as three growers have increased their plantings since the 1952-53 season and others indicate similar intentions for the coming season.

Table 1. Phony peach eradication in Missouri.

Year	Number counties		Number trees	
	Inspected	With phony peach	Inspected	Diseased
1930	6	0	---[a]	0
1931	34	0	---[a]	0
1932	15	2	---[a]	20
1933	13	4	---[a]	31
1934	3	3	---[a]	3
1935	35	5	---[a]	38
1936	37	14	363,625	219
1937	44	6	126,370	68
1938	46	1	92,269	2
1939	14	3	42,888	11
1940	18	1	44,568	1
1941	7	4	45,093	28
1942	4	2	14,020	20
1943	4	2	2,788	14
1944	4	1	2,256	4
1945	2	1	1,621	1
1946	2	0	1,395	0
1947	1	1	1,713	1
1948	1	1	1,431	1
1949	1	1	43,227	28
1950	1	1	36,733	1
1951	1	1	36,251	3
1952	1	1	38,605	5
1953	1	1	35,026	11

[a]Not available.

[1] Plant Pathologist, Department of Horticulture, University of Missouri.

[2] State Entomologist, Missouri State Department of Agriculture.

[3] Reported by John B. Williams, Bureau of Plant Industry.

Table 1 lists the number of counties inspected, the number of counties with infected trees, the number of trees inspected, and the number of trees infected.

Summary

The results of 24 years of inspection for and removal of phony-peach infected peach trees in Missouri indicate that the spread of the phony peach virus can be economically controlled. At the present time all but one county have been removed from quarantine list and the low annual infection found in this county emphasizes the value of this program.

UNIVERSITY OF MISSOURI AND MISSOURI STATE DEPARTMENT OF AGRICULTURE

VIRUS DISEASES OF SMALL GRAINS IN WYOMING

H. J. Walters

Three virus diseases which affect small grains have been found in Wyoming. These are wheat streak mosaic, barley false stripe, and barley yellow dwarf. Of the three viruses, wheat streak mosaic is most important.

Wheat Streak Mosaic Virus

Wheat streak mosaic was first recognized in the State in July 1951, but according to symptoms described by growers, it seems likely that the disease affected wheat in certain areas several years earlier.

Surveys in 1952 revealed wheat streak mosaic to occur in amounts varying from a trace to 100 percent in winter wheat in Laramie, Goshen, Platte, Niobrara, Sheridan, and Campbell Counties. Yield losses ranged from 10 to 100 percent in many fields in Laramie, Goshen and Platte Counties. The most severe infection and greatest losses occurred in areas where hail the previous year "threshed" mature grain onto the ground. Some loss in spring wheat occurred where it was planted near infected winter wheat.

By June 1953, wheat streak mosaic had spread to four other counties, when in addition to the six counties with known infestations in 1952, it was found in Johnson, Crook, Weston, and Carbon Counties. Infection ranged from a trace to 100 percent. Greatest losses occurred in southeastern Wyoming where 100 percent of 145 fields inspected contained mosaic. Several fields were not harvested because of the disease.

Surveys made in the fall of 1953 in southeastern Wyoming revealed wheat streak mosaic to be generally present in young winter wheat in Laramie, Goshen, and Platte Counties. Approximately 90 percent of 60 fields checked contained the disease. Heaviest infection occurred in volunteer and early-planted wheat. Infection was relatively low in late-planted fields, but in early-planted fields, as many as 100 percent of the plants were infected.

Where mature wheat was damaged by hail and in uncultivated fallow strips much volunteer wheat was heavily infected. The virus is not only spread from these areas to the fall-planted wheat but serves as a source for spring infection.

The warm late fall of 1953 and the open winter were conducive to a build-up in the population of the eriophyid mite, Aceria tulipae, the important vector of wheat streak mosaic virus, which resulted in severe infection of winter wheat as observed in June 1954. The disease was generally present over the eastern part of the State where winter wheat is grown, and crop loss was greatest in this area. In Goshen County many fields were plowed under in early June and planted to sorghums. Considerable acreage was not harvested in Platte County because of the disease. In northeastern Wyoming infection ranged from a trace to 25 percent. A few fields in Crook County showed 100 percent infection, with total loss in a few cases. Spring wheat infection was relatively high where grain was planted late and near infected winter wheat. A trace of infection was found in only a few fields of barley and oats.

Four annual grasses -- green foxtail (Setaria viridis), downy bromegrass (Bromus tectorum), Japanese bromegrass (B. japonicus), and barnyard grass (Echinochloa crus-galli) -- were found to be infected under natural conditions. Several perennial grasses present in the State are susceptible but have not been found to be naturally infected.

Wheat streak mosaic was found rarely in spring grains in the irrigated farming areas.

False Stripe Virus

False stripe, which was previously thought to be of nonparasitic origin, was observed in Frontier barley in the nursery at Laramie in 1953. In 1954, it was found at a rather high rate of infection in the Frontier, Compana, Spartan, and Glacier varieties of barley and in Pilot wheat in nurseries at Laramie, Torrington, Sheridan, and Gillette. Surveys revealed false stripe to be present in nearly all barley fields in northeastern Wyoming. In most fields infection ranged from a trace to 5 percent, but in one field infection was approximately 25 percent. In northcentral Wyoming only a trace of the disease was observed in a few scattered fields of barley. It was not detected in fields of any small grains in other parts of the State.

Yellow Dwarf Virus

In 1954 yellow dwarf was found throughout the State where small grains are grown. This disease has evidently been present for some time, but its presence had been attributed to non-parasitic causes and had not received much attention.

Losses are difficult to estimate, particularly in winter wheat where it is sometimes confused with wheat streak mosaic. Yellow dwarf virus was transmitted to Victory oats by two species of aphids, Macrosiphum granarium and Toxoptera graminum, from diseased plants of Cheyenne wheat collected in fields heavily infected with wheat streak mosaic virus.

At least a trace of yellow dwarf was present in nearly all fields of barley and oats inspected throughout the State. Heaviest infection appeared to be in the irrigated areas of Goshen County. In barley, heaviest infection was about 10 percent. The disease is more readily seen in oats owing to the red coloration of the oat leaves, and the incidence in oats appeared much higher than in barley. Aphid transmission of yellow dwarf virus from plants of barley and oats collected in the field was successful in the greenhouse.

Other Viruses

The three viruses discussed above have been definitely established as affecting small grains in Wyoming. Other viruses from eight collections of naturally infected wheat and downy bromegrass were not identified.

Inoculations made from part of these collections resulted in systemic symptoms on wheat and rye (Secale cereale), but not on foxtail millet (Setaria italica), or tall wheatgrass (Agropyron elongatum). The others induced systemic symptoms on wheat and foxtail millet but not on rye or tall wheatgrass.

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MOSAIC REACTION AND GEOGRAPHIC ORIGIN OF ACCESSIONS OF CUCUMIS MELO L.

Thomas W. Whitaker and G. W. Bohn[1]

Summary

Approximately 700 accessions of Cucumis melo were exposed to cantaloupe mosaic in the Imperial Valley of California during 1948-52 inclusive, under conditions where natural infection was estimated to be 100 percent.

Tolerance of and/or resistance to cantaloupe mosaic was confined principally to the botanical variety conomon. While not all tested accessions of conomon proved tolerant of or resistant to the disease, 78 percent of the 23 items considered useful for further breeding were conomon or conomon-like types.

The data indicate that the best sources of resistant plants are China and neighboring Korea, followed by northern India and Pakistan. Relatively large samples from Iran, Afghanistan, and Turkey, and smaller samples from Russia, western Europe, Africa, Iraq, Syria, Saudi Arabia, and South America showed little tolerance of or resistance to the disease.

During a systematic attempt to find material resistant to or tolerant of cantaloupe mosaic for use in a breeding program, about 700 accessions of Cucumis melo L. have been tested for their reaction to this disease. Seed of these accessions was secured for the most part through the cooperation of the Plant Introduction Section, Horticultural Crops Research Branch, U. S. Department of Agriculture. About two dozen accessions supplied by that Section tested over a 2- to 3-year period showed some tolerance of or resistance to the disease.

The results of these tests are of value because they demonstrate a more or less consistent disease reaction for each accession from year to year. Furthermore, the geographic origin and the botanical variety of the tolerant or resistant accessions suggest a pattern that may have significance. These observations should be of importance to muskmelon breeders, and to plant explorers searching for useful material of Cucumis melo for breeding purposes.

At least three viruses are involved in the cantaloupe mosaic complex (3). They produce similar symptoms on muskmelon, but can be separated by the symptoms they produce on three differential hosts, Cucurbita pepo L., Citrullus vulgaris Schrad., and Vigna sinensis (Torner) Savi. The relative importance of these viruses probably varies from year to year.

The reaction of the various accessions to the several specific viruses have not been determined. In this work we have depended entirely upon natural field infection. During the five seasons for which the data are recorded (1948 to 1952), infection was estimated to be 100 percent.

The scale suggested by Bohn and Whitaker (2) for rating the severity of the disease according to the symptoms expressed was used. For convenience in assessing the values given in Table 1, the scale is reproduced below.

Class	Leaf chlorosis	Leaf distortion and size reduction	Plant vigor
4.0	mild	trace	excellent
3.5	moderate	moderate	good
3.0	severe	severe	poor
2.5	severe	severe	poor
2.0	severe	very severe	very poor

Mosaic ratings were assigned to entire plots of the various collections. The plots were 15 ft. long, consisting of five hills spaced 2 1/2 feet apart and planted two vines per hill. For the most part, mosaic symptoms in the individual plots were suprisingly uniform. However, in some plots an occasional vine rated noticeably below or above the plot rating.

The range of the ratings of individual progenies from year to year was extremely narrow, suggesting that they were relatively homozygous for reaction to the disease. The evidence also

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Table 1. Reaction of 23 accessions of Cucumis melo of various geographic origins and of four commercial varieties to cantaloupe mosaic in the field.

P. I. accession No. or variety	Geographic origin	Number of tests	Mean mosaic rating
*132253	China	3	3.7
156928	China	3	3.0
*157069	China	5	3.6
*157070	China	6	3.8
*157071	China	7	3.3
*157073	China	8	3.4
157075	China	2	3.0
*157076	China	8	3.3
*157079	China	8	3.3
*157080	China	10	3.6
*157081	China	14	3.6
*157082	China	8	3.7
*157083	China	2	3.0
*157084	China	2	3.0
*157085	China	6	3.2
*157086	China	5	3.1
*158756	China	4	3.4
*161375	Korea	2	4.2
163208	Pakistan	3	3.2
*164323	India	3	3.3
164330	India	3	3.2
*164487	India	3	3.2
164584	India	3	3.3
Sulphur-Resistant V-1		3	3.2
Powdery Mildew-Resistant No. 5		4	2.8
Powdery Mildew-Resistant No. 45		3	2.7
Melogold		3	2.0

*Accessions that correspond to Cucumis melo var. conomon.

indicates that within limits environmental factors did not seriously disturb the ratings.

In Table 1 are recorded the number of tests and the mean ratings of 23 accessions considered to have some tolerance of or resistance to mosaic, along with those of 4 commercial varieties for comparison. The first time an accession was tested the original seed was used. Thereafter, it was represented by either or both self-pollinated or open-pollinated selections from the original material. For a high degree of reliability, two, and preferably three, tests were required.

In our analysis of the 23 collections recorded in Table 1, 18 (132253; 157069; 157070; 157071; 157073; 157076; 157079; 157080; 157081; 157082; 157083; 157084; 157085; 157086; 158756; 161375; 164323; and 164487) correspond to Cucumis melo var. conomon (Thunb.) Makino (Oriental Pickling Melon) as described by Bailey (1). In general, these accessions are characterized by extremely vigorous, prolific vines with heavy dark-green foliage. Leaves and stems are covered with short stiff bristles (setose), making the vines harsh to the touch. The fruits are small (3 to 4 in. long, 1 1/2 to 2 in. diameter), usually oblong, smooth; mostly without pronounced sutures, with light-green, yellow-green or yellow skin and thin white or green flesh; the cavity is entirely filled with placenta; the seeds are small, white or creamcolored. The flesh is tasteless and inedible.

Of approximately 700 accessions obtained from the Plant Introduction Section, 37 percent

were from Iran, 19 percent from Afghanistan, 14 percent from India and Pakistan, 11 percent from Turkey, 7 percent from China, 6 percent from Russia, and 4 percent from western Europe, and the remaining 2 percent included three from Africa, three from South America, two from Korea and single accessions from Iraq, Japan, Syria and Saudi Arabia.

The 23 accessions considered to have superior tolerance or resistance originated as follows: 74 percent from China, 22 percent from India and Pakistan, and 4 percent from Korea. It appears that the best sources of materials tolerant of or resistant to cantaloupe mosaic are China and neighboring Korea. The accessions from China originated in the north-western provinces Kansu and Shensi, the central provinces Hupeh, Chekiang and Szechwan, and the southern province Kwangsi. Most of the remaining accessions originated in northern India.

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VEGETABLE CROPS SECTION, HORTICULTURAL CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, U. S. DEPARTMENT OF AGRICULTURE

THE SPREAD OF CUCUMBER MOSAIC VIRUS IN CUCUMBER PLANTINGS DURING HARVESTING[1]

C. R. Amen and C. A. Porter[2]

Summary

Field experiments conducted at Corvallis, Oregon, during 1951 and 1952 to determine the importance of harvesting operations in the dissemination of cucumber mosaic virus within cucumber plantings showed that harvesting can be the major factor responsible for disease spread if diseased plants are initially present in the plantings.

For several years the spreading of cucumber mosaic virus within fields of pickling-cucumbers has caused large losses in Oregon. That this spread is not principally due to insect vectors is indicated by the following observations: (a) the spread has not correlated with the presence and distribution of vectors; (b) the spread has been more rapid after picking began, a period when vectors were scarce.

Doolittle[3] and Doolittle and Walker[4] state that thinning, training, and picking are responsible for some transmission of cucumber mosaic virus in the field, but that these means of dissemination are secondary to spread by insect vectors. In 1951 and 1952 experiments were conducted at Corvallis, Oregon, to determine to what extent the picking operation could spread the disease in the fields. These experiments showed that picking can be a major factor responsible for virus spread if diseased plants are present in the fields.

Methods

During the 1951 and 1952 seasons 16 rows of Snow's Perfection cucumbers were planted in a field at a college experimental farm near Corvallis, Oregon. The rows were 4 feet apart and 50 feet long. Two rows at each edge of the planting were not inoculated but were picked all during the season. These rows served as buffers and control plots. Each of the remaining 12 rows was considered a separate plot and every 2 rows as a block. Thus, in every block there was a row that was picked throughout the season and one that was left unpicked.

In 1951 an attempt was made on August 4 to inoculate every tenth plant with juice from a tobacco plant infected with cucumber mosaic virus. Not all of the inoculated plants became infected. Those rows in which no plants became infected after inoculation were considered controls at the end of the season.

In 1952, 3 plants at each of 3 locations in each row were inoculated on July 22 with juice from a diseased tobacco plant. After symptoms began to appear 2 of the 3 treated plants were rogued from each treatment location. This left 1 diseased plant at each location and 3 diseased plants in each row.

Each year the plantings were made late in June so that the plants did not emerge until after the major part of the spring aphid migration had passed. After the plants emerged they were dusted with 1% parathion at weekly intervals until picking started. In this way introduction of virus into the field by insects was kept as low as possible. Picking was begun August 1 each year. The plots were picked twice a week until disease readings were made in the middle of September. Pickers washed their hands carefully with soap and water after picking each row so that virus from one row would not be carried to another. Disease counts were made by pulling all plants in each plot and examining each plant individually for symptoms.

Discussion

In 1951, as shown in Table 1, plots not inoculated but picked had no diseased plants all season. Plots that contained inoculated plants and were picked 12 times had an average of 36.9

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[1] Approved as Technical Paper No. 880 by the Director of the Oregon Agricultural Experiment Station. Contribution of the Departments of Entomology and Botany and Plant Pathology.

[2] Present address: Boyce Thompson Institute, Yonkers, N. Y.

[3] Doolittle, S. P. 1920. The mosaic disease of cucurbits. U. S. Dept. Agr. Bull. 879: 69 pp.

[4] _____, and M. N. Walker. 1926. Control of cucumber mosaic by eradication of wild host plants. U. S. Dept. Agr. Bull. 146: 15 pp.

Table 1. 1951 transmission of cucumber mosaic by pickers in experiments at Corvallis, Oregon.

Treatment	Replicates	Average number of plants per replicate					
		Total plants	Infected inoculates	Total diseased		Disease increase	
				Number	Percent	Number	Percent
Inoculated and picked	3	40.7	3.0	15.0	36.9	12.0	29.5
Inoculated not picked	4	40.5	2.75	6.0	14.8	3.25	8.0
Not inoculated picked	7	42.7	0	0	0	0	0

Table 2. 1952 transmission of cucumber mosaic by pickers in experiments at Corvallis, Oregon.

Treatment	Replicates	Average number of plants per replicate					
		Total plants	Infected inoculates	Total diseased		Disease increase	
				Number	Percent	Number	Percent
Inoculated and picked	6	45.3	3.0	35.7	78.8	32.7	72.2
Inoculated not picked	6	39.5	3.0	12.8	32.4	9.8	24.8
Not inoculated picked	4	50.7	0	2.0	3.9	2.0	3.9

percent of the plants diseased in each plot, while the inoculated plots that were not picked contained an average of 14.8 percent diseased plants. The absence of diseased plants in the picked control plots indicated that no virus was transmitted by insect vectors during the season. The spread of virus in the unpicked plots was believed to be caused by the training of the vines in the rows. Training was necessary to prevent runners of plants in unpicked plots from contacting vines in the plots that were being picked.

Table 2 shows the extent of virus spread in the 1952 plots. Uninoculated plots picked 12 times had an average of 3.9 percent of the plants diseased at the end of the season. Plots that were inoculated and picked contained an average of 78.8 percent, and unpicked inoculated plots an average of 32.4 percent, diseased plants.

Transmission in the uninoculated picked rows in 1952 was probably accomplished in part by insect vectors that were not controlled by the weekly dust applications and in part by pickers going through these plots after a plant had been infected by natural means. As in the 1951 plots, some of the spread of virus in the unpicked plots was possibly caused by the necessity of training the vines in the rows.

The differences in the amount of mosaic spread between the two years might be explained by the different dates when the virus-source plants were inoculated. Mechanical inoculations in 1951 were made two weeks later than in 1952. The first and second pickings in 1951 were made before the plants were inoculated and the third and fourth pickings were made before the inoculated plants had become systemically infected. All pickings in 1952 were made after the virus had become systemic in the inoculated plants. Therefore, the opportunity for spread of the virus to new plants during the first four and subsequent pickings was much greater in 1952 than in 1951.

These experiments show that pickers are able to spread virus to a high percentage of cucumber plants in a field if a source of virus is initially present. Commercial cucumber growers in Oregon plant their crop from the middle of May to early June. The peak migration of winged aphids known to transmit cucumber mosaic occurs in June and early July when the cucumber plants are emerging from the soil. Since winged aphids continually enter and leave the cucumber fields at this time, it is believed that even frequent insecticide applications would not prevent some mosaic from being brought into the plantings. The virus brought into the fields by the migrating aphids would provide a source for further spread by pickers during harvest.

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THE INTERRELATED EFFECTS OF BIG VEIN AND MARKET PRICE ON THE YIELD OF HEAD LETTUCE

F. W. Zink and R. G. Grogan

Chupp and Paddock[1] reported that sporadic losses from the big-vein virus disease of lettuce in New York State ranged from 10 percent to so nearly total that some of the fields were plowed under before harvest time. The syndrome they described included necrotic stipples, spots and blotches, and if infection occurred early enough and was severe enough the plants oftentimes died.

Although big vein occurs every year, especially in the spring crop of the central coastal area of California where practically 100 percent of the plants in some fields are affected, it usually has not been considered a serious factor in limiting production.

The symptoms of big vein of lettuce under California conditions agree closely with the original description by Jagger and Chandler[2]. Affected plants do not develop any necrosis, but assume a decidedly upright habit of growth and the veins become slightly cleared. The vein clearing gradually becomes more intense and the leaves pucker slightly, giving the margins a ruffled appearance. The interveinal tissue adjacent to the veins also becomes translucent giving the illusion that the veins are enlarged. Observations, but without data needed for substantiation, indicate that head size is reduced and maturity is delayed. Severely affected plants often fail to head. Quality is also lowered in that heads from affected plants appear coarse, ribby and generally unattractive.

Even though big-vein-affected plants have many undesirable characteristics, growers and shippers often question whether or not the disease causes an economic loss. As an argument to support this viewpoint they can relate instances in which fields almost 100 percent affected with big-vein yielded well and returned a good profit; in fact, often more profit than predominantly healthy fields harvested at other times under less favorable market conditions.

The present study was made to determine what effect, if any, big vein exerts on yield of marketable lettuce. Since profit or loss to the grower is determined largely by market price, all data were considered in view of the market conditions which prevailed at the time of harvest.

Procedure

Ten commercial Great Lakes lettuce fields which had various percentages of big vein were selected for study. Care was taken to select fields with only traces of other lettuce diseases such as the common lettuce mosaic or aster yellows virus diseases. Harvest data were recorded from 5 100-foot sections of bed[3] in each of the 10 fields. Just prior to the first cutting (made by regular field crews) the plants that were showing big-vein symptoms and those which were apparently healthy were counted. After each cutting counts were made of the big-vein and healthy plants that had not been harvested. By calculation the percentages of the big-vein and clean populations harvested at each cutting and after 2 or 3 cuttings were determined.

On the basis of the data presented in Table 1, the effect of big vein on yield of head lettuce can be summarized as follows:

1. A large percentage of the healthy population as shown under "total percentage harvested" was cut in all trials regardless of market conditions. This is emphasized in that 87 percent of the total healthy population was cut while only 48 percent of the total big-vein population was harvested.
2. A heavy cut-out was made from predominantly healthy fields regardless of market condition. For example, 88 and 85 percent of plots 2 and 4 was cut during a good market and 84 percent of plot 10 during a poor market period.
3. In fields with predominantly high percentages of big-vein-affected plants, an unusually high percentage of the healthy population was harvested in the first cutting. For example, 90 percent of the healthy population of plots 8 and 9 was harvested in the first cutting. This indi-

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1 Chupp, C., and W. C. Paddock. 1949. Big vein of lettuce in New York State. Plant Dis. Reptr. 33: 280-281.

2 Jagger, I. C., and N. Chandler. 1934. Big vein, a disease of lettuce. Phytopath. 24: 1253-1256.

3 A lettuce bed in the Salinas district is a raised ridge 20 inches across the top on which 2 rows are planted 14 inches apart. The plants in each row are thinned to intervals of about 1 foot.

Table 1. The effect of big vein on yield of Great Lakes Lettuce in 10 commercial fields in the Salinas Valley.

Plot	Total plants	Prior to harvest percentage		Percentage of big-vein and clean populations harvested at each cutting						Total percentage harvested		Total percentage of all plants harvested	Market conditions* (price in dollars)
				1st cutting		2nd cutting		3rd cutting					
		Clean	B.V.	Clean	B.V.	Clean	B.V.	Clean	B.V.	Clean	B.V.		
1	893	57	43	11	0	35	14	25	18	71	32	55	1.50-1.75, mostly 1.50
2	888	91	9	28	5	42	12	24	11	94	28	88	1.50-2.00, mostly 1.75
3	842	36	64	18	6	45	11	15	16	78	33	49	1.50-2.00, mostly 1.75
4	863	77	23	44	8	45	10	8	2	98	38	85	1.50-2.00, mostly 1.75
5	850	39	61	41	19	51	40			92	59	72	1.75-2.25, mostly 2.00-2.25
6	857	40	60	27	8	41	12	5	21	73	41	54	1.50-2.00, mostly 2.00
7	833	34	66	5	0	40	22	36	33	80	55	64	2.25-3.00, mostly 2.50-2.75
8	849	5	95	90	18	7	41	0	15	97	74	75	2.50-3.10, mostly 3.00
9	869	11	89	90	11	5	26			95	37	43	1.00-1.10, mostly 1.00
10	835	94	6	18	6	33	16	37	4	88	25	84	1.00-1.25, mostly 1.10-1.15
Total for all plots		49	51	27	10	39	24	20	13	87	48	67	

*F.O.B. Salinas price for 2 doz. carton pack. $1.00-1.25 market poor, $1.25-1.50 market weak, $1.50-2.00 market good, $2.00 and up strong.

cates that big-vein delays maturity. Time for beginning of harvest in these two fields probably was judged on the basis of maturity of the big-vein population. Consequently when the first cutting was made practically all of the healthy population was mature or overmature and therefore was harvested at the first cutting.

4. Fields with high percentages of big-vein-affected plants yielded low cut-out percentages except when cut during a strong market period. This is illustrated by a comparison of plots 1, 3, and 9, with cut-out percentages of 55, 49, and 43, respectively, on a poor market, while plots 5 and 8 with as great or greater percentages of big-vein cut-out 72 and 75 percent, respectively, on a strong market.

Discussion

The data presented in Table 1 show that big vein affects yield, but not in a straight-line relationship correlated with percentage infection as is usually true of a disease with a syndrome that renders the lettuce completely unmarketable. Big vein exerts its greatest effect on yield during weak market periods. It is axiomatic that the best quality product from the standpoint of correct size for a certain pack and freedom from defects is shipped when the market is poor and that a product of lesser quality is also acceptable when the market is good. This is understandable in that a short supply of lettuce results in a good market or, in other words, increased demand for any type of lettuce. As a result undersized heads of poorer quality are accepted by buyers when the market is good but would be refused if an adequate supply of good quality lettuce were available.

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BROWN SPOT COMPLEX OF HEAD LETTUCE ON EASTERN MARKETS

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Abstract

Brown spot of head lettuce found on eastern markets is apparently not a specific disease entity but a complex of symptoms. The breakdown is probably physiological in nature and may develop in response to a number of different adverse conditions in the field or while the lettuce is in transit or storage. One or more of the symptoms resemble those of previously described lettuce diseases, especially russet, brown blight, spotted wilt, rib blight, redheart, and internal breakdown. In view of the practical difficulty in differentiating the various types of brown discolorations of lettuce on the market the name brown spot is proposed for those disorders whose cause is not definitely known or cannot be determined under market conditions. While there is some similarity of the brown spot complex to tipburn, it is believed that these lettuce disorders are not the same since they apparently develop in response to different environmental conditions.

From about the end of March to the middle of June, 1953, samples of discolored head lettuce (Lactuca sativa var. capitata L.) from widely separated lettuce-growing districts of the United States were received for diagnosis. Regardless of the origin of the lettuce, the symptoms on the affected heads were somewhat similar, being characterized by brown spotting of the leaves. Since a high percentage of the lettuce in some carlot shipments was reported affected and the nature of the trouble was in question, some work on the problem was undertaken.

Description of the Brown Spot Complex

During the observations it became apparent that at least three types of brown spot were present in lettuce on eastern markets: namely, a rib discoloration of field origin, a post-harvest breakdown, and a necrosis associated with decay. In slightly affected heads it was usually possible to differentiate these. In more severely affected heads, however, the symptoms of the three types of brown spot, which in some instances may possibly be different phases of one condition, might occur simultaneously on individual leaves and it was not possible to determine the type or origin of the breakdown.

Rib Discoloration of Field Origin: In the initial stages there is a small, diffuse, light-brownish discoloration of the midribs of one or a few of the outer head or wrapper leaves (Fig. 1A). In later stages the midribs of the inner leaves also are affected, the discoloration extends into the larger and smaller veins, and finally the interveinal tissues become discolored. At the late stages the affected tissues usually are sunken and pitted. At first the lesions are light in color (tilleul-buff, shrimp pink, or light ochraceous-salmon), later the discolorations darken (orange-citrine and russet), and in the final stages of the disorder the lesions are dark (army brown to blackish brown)[2]. The discoloration usually starts at the region of greatest curvature of the rather prominent midribs and is more intense on the inner (upper) side of the leaves. Often the tissue on the outer (lower) side of the midveins is somewhat shrivelled or puckered, and the midribs are usually spongy with a cavity on each side of the main vascular bundle. Microscopic examination of free-hand cross-sections of the ribs and veins reveals that the initial stages of the discoloration are the result of a browning of the mesophyll cells on the sides of the vascular bundles as well as those adjacent to the midrib cavities. In later stages the discolored mesophyll cells are connected on the upper side of the bundles by a line of discolored and collapsed or disorganized cells that form an irregularly crescent-shaped area. In addition, irregular fissures may be present in the mesophyll tissue of the ribs and veins, and the discoloration extends into the epidermal and xylem cells as well as into the lactiferous ducts.

In the initial stages there is no evidence of bacteria or fungi, but later masses of bacteria are present in the larger spots, especially in the pitted ones, and when conditions of temperature and humidity are favorable a slimy soft rot develops very rapidly. The leaf tissue in the advanced stage is firm and rubbery or parchment-like to the touch but not soft, and there is no

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[1] Plant Pathologist, Biological Sciences Branch, Agricultural Marketing Service, U. S. Department of Agriculture, New York, New York.

[2] Colors according to Ridgway (23).

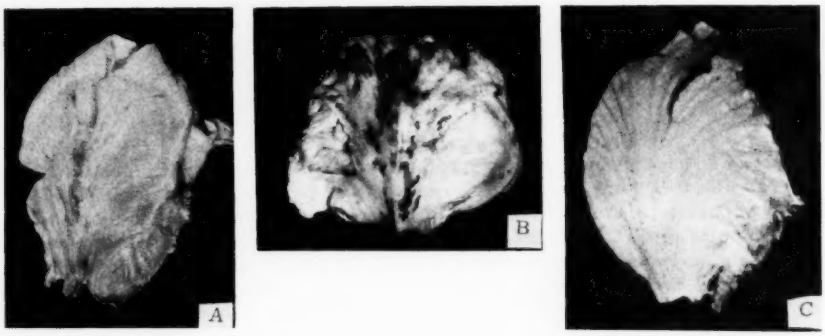

FIGURE 1. Lettuce brown spot. A --Brown spot of field origin (rib breakdown type). B -- Interveinal type of brown spot occurring in transit and storage. C -- Brown spot on inner leaf from head of lettuce that had extensive bacterial soft rot on outer leaves.

microscopic evidence of dissolution of the middle lamella of the disorganized and shrivelled cells as in those affected with bacterial soft rot.

Brown spot (rib discoloration) was observed in samples of lettuce from commercial shipments from California (Imperial and Salinas Valleys), Arizona, Texas (Uvalde section), Florida, Georgia, South Carolina, and Virginia.

Post-Harvest Breakdown: In a number of samples from carload lots of western-grown lettuce shipped to eastern markets, a post-harvest breakdown has been observed. The most prominent symptom is a pronounced brown spotting of the interveinal tissue of the inner head leaves while little midrib or vein discoloration is observed in early stages of the disorder. In very severely discolored heads the outer head leaves and occasionally the wrapper leaves are affected (Fig. 1 B). Except for the appearance of the earliest symptoms on the inner head leaves and the interveinal tissue, this internal form of brown spot is similar macroscopically and microscopically to brown spot described in the preceding section.

A similar type of brown spotting of the inner leaves, often called storage breakdown, was observed in the spring of 1953 in New York City in western-grown lettuce stored in a sound condition for 2 to 4 weeks at 32° to 38° F.

A breakdown of the inner leaves, superficially resembling the internal type of brown spot, was observed on lettuce packaged in non-perforated pliofilm bags in which the oxygen content of the air had fallen as low as 3.5 percent. The heads from such packages had a sour odor, and the inner leaves were water soaked and translucent and had a sickly brownish-gray cast. Brown spots were not found on the ribs, veins or interveinal tissue, and no cell browning, collapse, or bacteria were observed even when the entire leaf was affected.

Brown Spot Associated with Decay: Lettuce with pronounced slimy soft rot of the outer leaves is regularly found on the market. Stripping off the outer decayed leaves of occasional heads reveals a type of brown spot which occurs mainly on the inner head leaves (Fig. 1C). These lesions appear similar to the symptoms of brown spot described previously; but they differ microscopically as the discoloration starts in the epidermal cells of either surface of the leaf and progresses into the mesophyll tissue, and also there is considerable discoloration of the phloem and xylem cells of the vascular bundles at the time the epidermis is affected.

Relation of Brown Spot to Bacteria

Although in 1949 attempts to isolate a pathogenic organism from brown spot lesions had failed, efforts were renewed in 1953 as large masses of bacteria were seen in the later stages of the three types of brown spot. Thirty-seven cultures of white and yellow bacteria were isolated from affected tissues. Inoculation with these organisms failed to reproduce the disease. In a few instances standing the cut bases of lettuce leaves that had midrib cavities in water with or without the addition of bacteria caused midrib discoloration after 3 to 12 days in a moist

chamber at room temperature. The discolored midribs showed a typical browning of the mesophyll cells around the cavities, and in more advanced stages some cellular collapse and large masses of bacteria. However, sunken spots did not develop on the midribs, veins were not affected, and no brown spotting on the tissue between the veins was seen.

Association of Tipburn and Brown Spot

Both brown spot and tipburn were present in 4 of the 11 samples of lettuce received at the laboratory for diagnosis. As brown spot was reported[3] to develop under exactly the same conditions as tipburn did, it was decided to make a random sampling of certificates of lettuce inspections at several eastern markets from April 15 to June 3, 1953, to ascertain the frequency with which tipburn and brown spot were associated in market inspections. In 52 of 136 certificates examined neither disease was reported. Tipburn alone was reported in 52 certificates, brown spot alone in 19, and both diseases together in 13. The two diseases were therefore reported associated in 15 percent of the 84 affected lots of lettuce inspected.

Discussion

The type of brown spot most frequently found at the New York market during the spring of 1953 was the midrib and vein discoloration of field origin. This type of brown spot appears to be similar to or to resemble in one or more respects russet (17), vein browning (26), rust (18)[4], redheart found in the spring crop (20), a tipburn-like condition (8), an unnamed New Jersey disease (19), the affected lettuce seen on the Chicago and New York markets in 1949[5], a disease of lettuce associated with saline soils (28), and rib blight (4).

In addition, the symptoms of brown spot (rib breakdown type) and brown blight are markedly similar (14). The occurrence of both in toxic or saline soils has been observed by Bardin[6] and others (4, 28). Although brown blight was first observed about 1917 and believed to be infectious in nature, isolation of an etiologic agent from affected lettuce grown in the Far West has not been reported yet. A virus disease of lettuce reported to resemble both brown blight and spotted wilt has been observed in Indiana and North Carolina (2, 3, 24). The validity of brown blight as a specific disease entity has been questioned (5). It has been noted that the symptoms of brown blight and spotted wilt, when maturing heads of lettuce are attacked, are very similar (16), and these two diseases may have been confused occasionally on the market (21). In view of the resemblance of the symptoms of spotted wilt and the internal breakdown phase of aster yellows to brown spot[6], any reports of the first two diseases without definite identification of the causative viruses may be questioned.

Tipburn has been reported in the spring crop (4, 6, 7, 8) and in plantings where a brown spot-like condition also was found (19, 26). The early stages of breakdown in tipburn and brqwn spot appear to be caused by physiological conditions, while in later stages saprophytic bacteria invade affected tissue very rapidly under favorable conditions of temperature and humidity and cause slimy soft rots. It has been postulated (1, 10, 12, 15, 27, to cite a few reports), that tipburn, like brown spot, is favored by a wide variety of conditions. On the other hand, some lots of lettuce with brown spots had no tipburn[5], and tipburn and brown spot were found together in only 15 percent of 84 affected lettuce shipments during the spring of 1953. That the two diseases are not necessarily directly related may be inferred from some of the numerous descriptions of tipburn which make no mention of symptoms resembling brown spot (1, 5, 10, 22, 25, 27).

The post-harvest type of brown spot breakdown of lettuce appears to be physiological in nature and similar to redheart (when found in cars delayed in transit or after long storage at low temperatures) (20), internal browning (7), or storage breakdown (9). This internal type of brown spot in western-grown lettuce on eastern markets may possibly result from delay in transit or inadequate refrigeration en route, but in some cases adverse growing conditions when the heads of lettuce are maturing or the harvest of overmature heads may predispose the lettuce to brown spot breakdown in transit.

While the use of different wrapping materials was reported to have little or no effect on the

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[3] R. C. Thompson, personal communication, 1953.

[4] The term "rust" has been used occasionally to describe the symptoms caused by the lettuce-mosaic virus (13). The vein necrosis and interveinal necrotic lesions which develop occasionally in this disease are often difficult to distinguish from the early symptoms of spotted wilt (11).

[5] G. B. Ramsey and B. A. Friedman, unpublished results, 1949.

[6] R. Bardin, personal communication, 1953.

incidence of internal browning (7), Nelson (20) found it to develop under conditions of reduced oxygen supply. An apparently similar type of anaerobic breakdown observed at this laboratory did not resemble the brown spot that developed in transit or storage.

The type of brown spot associated with decay is probably similar to the type of redheart (20) with which extensive bacterial soft rot is associated. In the later stages of this epidermal type of breakdown numerous bacteria are present in the lesions, but whether the initial stages are physiological in origin or the result of incipient bacterial infections was not determined.

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BIOLOGICAL SCIENCES BRANCH, AGRICULTURAL MARKETING SERVICE, U. S. DEPARTMENT OF AGRICULTURE, NEW YORK, NEW YORK.

INCIDENCE OF TOBACCO BLACKSHANK DIRECTLY RELATED TO SOIL pH[1]

Randall R. Kincaid and Nathan Gammon, Jr.[2]

ABSTRACT

The soil of shade-grown cigar-wrapper tobacco plots was adjusted with elemental sulfur and hydrated lime to five pH levels, from about 4.3 to 6.4. Incidence of blackshank in a susceptible variety increased with each increment of pH until near the end of the season, when mortality was complete or almost complete except at the lowest pH. Correlation between blackshank and pH was higher than between blackshank and exchangeable calcium, but both were highly significant. Incidence of blackshank in a resistant variety was low, but showed the same trend.

Incidence of blackshank (Phytophthora parasitica var. nicotianae (Breda de Haan) Tucker) varied directly and significantly with soil pH, according to unpublished results obtained at Quincy, Florida, in 1951 and 1952, in experiments designed for other purposes. Irvine and Valleau (1) reported, "Studies on ... pH suggest that fields on which the pH concentration is high (that is, 6.5 or above) are more likely to develop the disease and carry the fungus from year to year than soil more distinctly acid (pH 5.6 or below)." Valleau, Johnson, and Irvine (4) stated, "It seems probable that the presence of lime in the soil is more of a factor in persistence of the fungus than pH concentration."

This paper reports results of an experiment designed to test these relationships.

EXPERIMENTAL

The experiment was conducted on an area of blackshank-infested Marlboro fine sandy loam, where blackshank-resistant varieties of shade tobacco had been grown for the preceding four years. The area was divided into 20 plots for five treatments in quadruplicate randomized blocks. Dusting sulfur 93% or commercial hydrated lime was broadcast and then mixed with the soil 6 inches deep by means of a rotary tiller, December 18, 1953.

Blackshank-free seedlings were transplanted under shade cloth March 26, 1954. Two rows of each plot, one of which was fumigated with ethylene dibromide February 5, were transplanted in a resistant variety, Dixie Shade (3), and one row, unfumigated, in a susceptible variety, No. 870 (2). Mortality data, including plants either dead or severely stunted with blackshank, were taken at intervals of about a week. On July 2, shortly after the end of leaf harvest, plants of the the resistant variety were pulled and the roots were rated for blackshank injury; a blackshank index, based on a maximum of 100, was calculated for each row. Soil analyses for pH and exchangeable calcium were made for each row of the resistant variety. Since no important differences were found between fumigated and unfumigated rows in this experiment, soil analyses and blackshank results for the resistant variety were averaged.

RESULTS

Results, given in Table 1, show that the incidence of blackshank in the susceptible variety increased with each increment of pH until near the end of the season, when mortality was complete or almost complete except at the lowest pH. Coefficients of correlation[3], using results of all 20 individual plots, were as follows: Mortality April 26 and pH April 23, $=+.81 \pm .05$; mortality April 26 and replaceable calcium (CaO) April 23, $=+.64 \pm .09$. Correlation with pH gave the higher coefficient, but both were highly significant.

Mortality and blackshank index in the resistant variety were low, but showed the same trend.

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1 Florida Agricultural Experiment Station Journal Series, No. 308.

2 Plant Pathologist, North Florida Experiment Station, Quincy, Florida, and Soils Chemist, Agricultural Experiment Station, Gainesville, Florida.

3 Calculated by methods given by Hayes and Garber, Breeding Crop Plants, 2nd ed. New York: McGraw-Hill. 1927.

Table 1. Incidence of blackshank of tobacco at five pH levels, 1954.

Treatment No.	Soil treatment: Material	Rate lbs./A.	Soil pH: March 2	April 23	July 13	Exchangeable Ca as CaO April 23 lbs./A.	Mortality of susceptible variety: April 26	May 25	June 26	Resistant variety: Mortality	Blackshank index
							%	%	%	%	
1	Dusting Sulfur	605	4.33	4.45	4.61	290	24	38	58	1	2
2	None	0	5.19	4.97	5.24	429	67	87	100	1	2
3	Hydrated Lime	908	5.66	5.42	5.51	691	74	92	95	2	6
4	Hydrated Lime	1,815	5.96	5.57	5.81	1,000	83	97	100	2	4
5	Hydrated Lime	2,723	6.40	6.05	6.17	1,500	88	99	100	3	6

DISCUSSION

The critical pH for blackshank suggested by field observations in Kentucky was about 5.6 (1). This was higher than that observed in the first year of the Florida experiment, which was 5.2 (pH of the unadjusted plots) or lower. It is possible that as the experiment is continued incidence of blackshank will decrease at the lowest pH and increase at the highest pH.

For breeding plots, where early and complete elimination of susceptible plants is desired, a soil pH somewhat higher than 5.2 is indicated. For commercial production of resistant cigar-wrapper tobacco in the vicinity of Quincy, Florida, the optimum pH may prove to depend largely on yield and quality factors, unless mortality is higher than observed in this experiment.

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FLORIDA AGRICULTURAL EXPERIMENT STATIONS

WATERMELON FRUIT ROT, CAUSED BY PHYTOPHTHORA PARASITICA, IN TEXAS

Don C. Norton and D. W. Rosberg

In June 1954, attention was called to a watermelon (Black Diamond) fruit rot (Fig. 1) in an irrigated field in the Lower Rio Grande Valley of Texas. Although growers stated that it had occurred in the past, the rot was most serious in 1954 when a 15 percent loss was sustained in some fields. Sporangia of Phytophthora were found on the surface of the lesions. Several isolations were made from the advancing margin of diseased tissues of many fruits, using potato-dextrose, malt, and corn-meal agars. The Phytophthora appeared consistently on all media. All isolates appeared similar in morphological and cultural characteristics on any given medium. All artificially inoculated melons became diseased. The fungus was identified as Phytophthora parasitica Dast. (4). Well grown tobacco plants were not killed until 25 to 30 days[1].

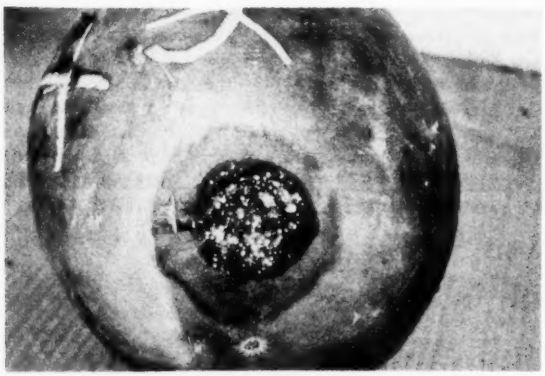

FIGURE 1. Watermelon fruit rot produced in the field by Phytophthora parasitica.

Watermelon fruit rots, in the field, have been reported to be caused by P. cactorum (Leb. & Cohn) Schroet. in Arizona (1), by P. capsici Leonian identified in New York on Winter Queen melons grown in Colorado (6), and by P. citrophthora (Sm. & Sm.) Leonian in California (2). Sherbakoff (5) produced a watermelon fruit rot in the laboratory by inoculation with P. parasitica. Other species of Phytophthora have also caused watermelon fruit rot by artificial inoculation. It is of interest to note the failure of P. palmivora Butler to incite a fruit rot of watermelon as reported by Malaguti (3).

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TEXAS AGRICULTURAL EXPERIMENT STATION, COLLEGE STATION, TEXAS

THIELAVIOPSIS BASICOLA ON CARROT ROOTS FROM CALIFORNIA

B. A. Friedman, W. R. Barger, and W. A. Radspinner[1]

In one of a series of tests to determine the effect of various types of refrigeration and containers on the shipping quality and shelf-life of carrots, two test packages, each containing 24 1-pound bags of topped carrots in perforated polyethylene, were received at New York City in August 1954.

Eight bags from each carton were examined at arrival, after a shipping period of 13 days during which the temperature averaged 45° F., and less than 1 percent of the carrots were found to have bacterial soft rot present. The remaining bags were stored at 69° F. Eight bags from each carton were examined after 3 days storage but no decay of any kind was found. At the end of 8 days storage at 69° the remaining carrots were inspected. About 13 percent of the fleshy roots had a black mold growth on their surface. The carrots had a few to many affected spots which were scattered indiscriminately and ranged in size from 3 to 20 mm. in diameter (Fig. 1). The mold usually affected only the skin but in some cases there was a dark gray discoloration of the underlying tissue to a depth of less than 1 mm. While peeling left most of the carrots edible, nevertheless the blemished appearance of the carrots before peeling rendered them commercially worthless.

FIGURE 1. California-grown carrots showing black mold lesions caused by Thielaviopsis basicola.

Examination showed the fungus to be Thielaviopsis basicola (Berk.) Ferraris[2]. There are apparently no previous reports of the presence of this mold on carrots. It is believed that the fungus was able to develop on this host because of the combination of high humidity within the polyethylene bags and the high temperature at which they were stored. In addition, carrot roots are apparently a favorable substrate for the growth of this fungus as Yarwood[3] has used living carrot slices for the isolation of Thielaviopsis basicola from soils and plant debris.

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[2] Acknowledgement is made to W. W. Diehl, Plant Disease Epidemics and Identification Section, Agricultural Research Service, U. S. Department of Agriculture for checking on the identification of the fungus.

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EFFECT OF SEED TREATMENT ON BEAN ROOT ROT

H. J. Walters

Root rots of beans cause much concern to growers in Wyoming, where the crop is grown under irrigation. A number of different organisms may cause these rots, but the principal ones are species of Fusarium, which normally inhabit practically all soils. These organisms attack the taproot and usually cause a reddish discoloration which may extend to the soil line. The main root and lower part of the stem become pithy and dry. Usually the rootlets that develop from the taproot are killed. When this occurs a cluster of rootlets develops above the lesion immediately below the soil line and keeps the plants alive.

Seed treatment tests were conducted with three varieties of beans to ascertain the effect of certain compounds on the development of root rot and resulting yields. In 1950, compounds used for seed treatment were applied as dusts, but results from such treatments were not satisfactory. From 1950 to 1953, methocel (methylcellulose) was used as a sticker for treatments with all fungicides except Dow 9B with which it is not compatible. Compounds were used at the following rates per 100 pounds of seed: Arasan--8 oz., Ceresan--2 oz., Spergon--8 oz., Dow 9B--2 oz., Dithane Z78--8 oz., and I & D (Arasan and Lindane)--3 oz.

Table 1. Root-rot index[a] of three varieties of beans after seed treatment with various fungicides.

Treatment	Great Northern UI[b] 16			Great Northern UI 123		Pinto UI 78		
	1950	1952	1953	1950	1951	1950	1951	1952
Arasan	71	53	51	64	35	61	51	44
Ceresan	71	--	--	75	32	61	31	--
Dithane Z78	56[c]	58	45	65	35	54	43	43
Captan	--	51	54	--	--	--	--	43
I & D	--	60	50	--	37	--	37	37
Check	70	58	57	67	43	61	36	42

[a] The higher the index number, the more severe the disease.

[b] UI refers to varieties developed by the University of Idaho.

[c] Underlined figures are significantly lower than check at the 5% point.

Treated beans were planted during the last week of May in soil heavily infested with root-rotting organisms. Each test consisted of four 25-foot rows replicated four times.

Root-rot readings were made during the second week in August. Ten plants were pulled at random from each of the outside rows of each plot, and the root system of each plant classified from 0 to 4 according to severity of disease. A disease index was prepared by multiplying the number of plants in each classification by the number assigned to that classification. After preparation of an index for each replication, the mean for the four replications was calculated. The index number is in direct correlation to severity of root rot: i.e., the higher the index number, the more severe the disease.

Yield data were taken from the two middle rows of each 4-row plot.

There was no direct correlation between treatment and severity of root rot or yield. The disease index of the treatments was significantly lower than that of the checks in only a few cases (Table 1). Also, severity of root rot is not correlated with yield. In several instances as good as or better yields were obtained from those treatments showing the highest index for root rot. None of the treatments resulted in increased yields significant at the 5% point over

Table 2. Pounds of beans produced per acre with three varieties of beans after seed treatment with various fungicides for control of root rot.

Treatment	Great Northern UI[a] 16			Great Northern UI 123		Pinto UI 78		
	1950	1952	1953	1950	1951	1950	1951	1952
Arasan	1650	1618	1918	1761	1672	1253	2047	1823
Ceresan	1478	--	--	1699	1601	1935	2232	--
Dithane Z78	1315	1561	1799	1749	1821	2232	2267	1975
Captan	--	1642	1856	--	--	--	--	2071
I & D	--	1561	1856	--	1268	--	1960	2251
Check	1674	1704	1856	1401	1601	1662	2047	2071

[a] UI refers to varieties developed by the University of Idaho.

the check (Table 2).

Results of treatments for one year, which included Spergon and Dow 9B, are not included in the tables. Severity of root rot was not significantly lower, nor were yields significantly higher than the checks.

Seed treatment with I & D in 1951 decreased yields, probably owing to too heavy application of the material which caused reduced stands and retardation of growth. Lighter applications of the fungicide were used in 1952 and 1953 and resulted in increased yields.

The various seed treatments did not show any definite control of the root-rotting organisms. Since Fusarium fungi are ever present in the soil and usually attack plants after they are past the seedling stage, not enough fungicide can be applied by treating seed to prevent infection of bean plants.

WYOMING AGRICULTURAL EXPERIMENT STATION, LARAMIE, WYOMING

EXPERIMENTAL CONTROL OF SOUTHERN BLIGHT ON TOMATO

P. A. Young

Sclerotium rolfsii is a rotting organism that causes southern blight of tomato and many other crop plants[1]. To try to control it with a chemical in 1954, metal-bordered plots, each 24 sq. ft., were built in a field where S. rolfsii killed 91 percent of the tomato plants in 1953. There were nine replications of the checks and of each chemical treatment, which included 3% and 4.5% chlorobromopropene (CBP). The 3% CBP was emulsified with Triton in 2 2/3 gallons of water and sprinkled on plots on March 29. Immediately thereafter, an equal amount of water was sprinkled on each plot to carry the chemical deeper into the soil. The 4.5% CBP was emulsified in 4 gallons of water and was followed by 4 gallons of water per plot. Each check plot was sprinkled with 5 1/3 gallons of water.

Only five weeds emerged in the 18 plots treated with CBP within a month, whereas 17,542 weed plants (Diodia teres and Digitaria sanguinalis) had to be pulled from the nine check plots.

On April 27, 32 plants of Manasota tomato were set in each plot. By July 1, none of the tomato plants in the plots treated with CBP showed any symptom of southern blight, whereas 18 percent of those in the check plots had died of the disease. The tomato plants were much larger in the plots treated with CBP. Southern blight killed 45 percent of the tomato plants in an adjacent row in this field. This adds to the data from a similar test in a nearby field in 1953[2].

Numerous tomato plants die of southern blight in hot dry weather in June to September when mycelium and sclerotia are not noticable on the crowns of the plants. In other parts of the field that contained the box-plots, designated Field A4D, most of the tomato crowns remained apparently healthy until southern blight had rotted the tap roots 2 to 8 inches deep. S. rolfsii may survive as mycelium in decaying crop materials or other organic matter in the soil.

Southern blight killed nearly all of the tomato plants in an experimental field, Field A1M in 1943. It was controlled by a 5-year rotation with rye and Bermuda grass. The disease killed 73 percent of the tomato plants in Field A4B, following corn in rotation, and 91 percent in Field A4D where tomato followed cotton in rotation, in 1953. Apparently corn and cotton had built up Sclerotium rolfsii in the soil of these fields without themselves showing recognizable symptoms in 1952.

Other methods of control are being studied. Many species, interspecific crosses, varieties, and hybrid selections of tomatoes were tested for six years at the Tomato Disease Laboratory for resistance to Sclerotium rolfsii. Nearly all of the inoculated tomato plants died of southern blight each year, but mild resistance was found in one interspecific cross, SR1252. A breeding-stock tomato with strong resistance to southern blight is being sought. Control with antibiotic organisms is unlikely in Norfolk sandy soil that contains only about 0.5 percent organic matter. Very deep plowing to bury all crop materials completely may control southern blight, but machinery for such plowing is unavailable. Very deep plowing would bring much infertile soil to the surface and probably would decrease crop yields for a few years.

Experimental control of southern blight obtained with CBP suggests that further tests should be made with this material to determine a method of commercial use.

TEXAS AGRICULTURAL EXPERIMENT STATION AND SHELL CHEMICAL COMPANY

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1 Young, P. A. 1946. Tomato diseases in Texas. Texas Agr. Exp. Sta. Circ. 113.

2 _____, and J. A. Pinckard. 1954. Sclerotium rolfsii and its control with chlorobromopropene. Phytopath. 44: 334.

AN ECONOMICAL CONTROL OF BLACK ROOT OF CAULIFLOWER

Leo Campbell

Black root (damping-off, wire stem) of cole crops, caused by Corticium solani (Pellicularia filamentosa) became so serious in western Washington that the development of an economical control of the disease was indicated. Since pentachloronitrobenzene (PCNB) gave such outstanding results in the control of club root of these crops without injuring the plants[1], it was tested against the black root disease of cauliflower.

PCNB was added at varying rates by two methods of application in three replicates of one 21-foot row each in strips 7 inches wide before the seed was drilled. The material was used at the rates of 50, 100, 150, and 0 pounds, rototilled into the soil to a depth of about two inches; and 10, 20, 30, and 0 pounds per acre applied as a soil-surface treatment. The rate of application in each case was in pounds actual per acre, broadcast equivalent.

Data on the results of these experiments are given in Table 1. An analysis of this data indicates that the results of the experiments were highly significant.

Table 1. Cauliflower black root control with PCNB at the indicated rates, rototilled into soil or applied as a soil-surface treatment. Number of plants (P) and number of diseased plants (D) per replicate, and totals, with average percent disease and average percent reduction in disease as compared with untreated checks.

Replicate	Rototilled Treatment -- lbs./A								Surface Treatment -- lbs./A							
	50		100		150		0		10		20		30		0	
	P	D	P	D	P	D	P	D	P	D	P	D	P	D	P	D
A	126	1	129	4	154	4	139	14	138	1	165	3	151	1	104	42
B	178	3	230	5	155	5	167	67	151	1	126	1	202	2	186	86
C	175	0	175	0	150	0	181	31	150	0	151	1	125	0	143	18
Total	479	4	534	9	459	9	487	112	439	2	442	5	478	3	433	146
Average % -- Disease	0.83		1.68		1.96		22.99		0.45		1.13		0.62		33.71	
Reduction	96.38		92.69		91.47		--		97.86		96.64		97.86		--	

Slightly better results were obtained where the fungicide was left on the surface of the soil, as might be expected, since infection occurs near the soil surface. None of the treatments reduced the stand or caused any other noticeable injury to the plants.

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1 Campbell, Leo. 1954. Fungicidal control of club root of cauliflower. Plant Dis. Reptr. 38: 283-285.

OBSERVATIONS ON THE OCCURRENCE AND CONTROL OF POWDERY MILDEW ON GREENHOUSE CUCUMBERS IN ONTARIO[1]

C. D. McKeen[2]

Abstract

Within the last decade in southern Ontario powdery mildew has become a serious disease of cucurbits, particularly of cucumbers grown under glass. The effectiveness of three fungicides for control of mildew on greenhouse cucumbers has been studied. Karathane (dinitro 1-methyl heptyl phenyl crotonate) has controlled powdery mildew more effectively than sulfur or copper sulfate. As a smoke, a form in which it is easily applied, Karathane is outstandingly effective.

In the last decade powdery mildew (Erysiphe cichoracearum DC.) has become a highly important disease of cucurbit (cucumber, muskmelon and squash) crops in southern Ontario. The disease has increased considerably in prevalence since 1944 when it was reported for the first time affecting a few crops of greenhouse cucumbers. In 1947 the disease reached epidemic levels in muskmelon fields in the Leamington area and reduced some yields by as much as 50 percent. Subsequently, growers have applied fungicides to reduce losses from the annual outbreaks of mildew in their field crops.

Since 1951, powdery mildew has become a highly destructive disease of cucumbers grown under glass in the important Leamington glasshouse area. Between 1944 and 1950, mildew occurred annually on a few crops during June and July, that is toward the end of the spring cropping season. A striking change was observed, however, when mildew appeared on cucumber seedlings in three different greenhouses in December of 1951. Mildew not only persisted in these greenhouses for the following six months, but at the same time, it served as the source of inoculum for other greenhouse cucumber crops in the area. This constituted the first overwintering of the fungus in the vegetative form in Ontario, and, as well, it demonstrated the destructiveness of the disease on the greenhouse cucumber crop. In 1953 and 1954 powdery mildew again appeared in the greenhouses and behaved as in the 1951-52 season.

The necessity for controlling powdery mildew, particularly on cucumbers grown under glass, led to a study of the most effective fungicides and the most satisfactory methods of applying them. The results obtained with sulfur, copper sulfate, and Karathane are presented here.

EXPERIMENTAL

(i) Sulfur: During the 1944-51 period, when mildew developed on greenhouse cucumber crops during the late spring and early summer, control was sought by dusting the plants lightly with finely ground sulfur. Although the sulfur caused appreciable hardening of the foliage and accompanying chlorosis, it controlled the mildew satisfactorily for a period of 4 or 5 weeks, or for as long a period as growers required for harvesting the greenhouse crop. Sprays containing wettable sulfur were less satisfactory because host injury was usually more severe than when the fungicide was applied in the dust form.

For mildew control the application of sulfur dust to greenhouse cucumbers during the winter months was unsatisfactory, chiefly for two reasons. First, even light dustings frequently caused too much necrosis of the tender foliage to permit its use. Secondly, when even slight plant injury was sustained, sulfur dust was not effective in holding mildew in check for more than 6 or 7 weeks, a period representing only one-quarter to one-third of the crop life. Eventually mildew infections developed profusely on the lower leaf surfaces and along the stems where the protective dust failed to adhere adequately. Consequently, sulfur dust proved unsatisfactory for mildew control when the disease appeared in greenhouses early in the winter.

(ii) Copper Sulfate: Yarwood (6) in evaluating the effectiveness of several fungicides

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[1] Contribution No. 1420, from the Botany and Plant Pathology Division, Science Service, Canada Department of Agriculture, Ottawa, Ontario.

[2] Senior Plant Pathologist.

against powdery mildew in California found that sprays containing 0.03 percent copper sulfate plus a spreader gave excellent control of mildew on cucumbers and beans. He reported that this treatment caused less host injury than did a light dusting with sulfur. The writer found that sprays containing 8 to 12 ounces of copper sulfate in 100 gallons of water plus a spreader destroyed the superficial mycelium of the fungus. However, because of the absence of residual protective action, satisfactory control of mildew was obtained only if sprays were applied regularly at 5- to 7-day intervals. After each spraying with copper sulfate, mild to moderate and, occasionally, even severe chlorosis and necrosis of the foliage resulted. Consequently, copper sulfate sprays were considered to be moderately safe only on mature crops.

The effectiveness of copper sulfate applied as an aerosol was also tested. For this method a microsol dispenser[3] which projected a mist of an alcoholic solution of copper sulfate throughout the greenhouse was used. Five treatments at 5- to 7-day intervals were made over a period of 28 days. Whereas the fungus was prevented from developing on the upper leaf surfaces for this period, mildew infections developed profusely on the lower leaf surfaces and to a lesser extent along the stems. Both adjustment of the particle size and efforts to maintain turbulence of the air around the plants for 3 or 4 minutes after application failed to improve control appreciably.

(iii) Karathane (dinitro 1-methyl heptyl phenyl crotonate)[4]: In 1945 Yarwood (6) reported Karathane to be the most effective of several fungicides tested for the control of powdery mildew on bean in California. Godfrey (1) reported that the same fungicide containing 1 percent active material in the dust form gave excellent control of powdery mildew on cantaloupes in Texas.

In tests conducted by the writer sprays containing 5 to 8 ounces of Iscothan (15 percent active material) in 100 gallons of water plus a spreader were applied to cucumber plants heavily infected with mildew. Within 36 hours mildew infections assumed a yellowish cast and subsequently failed to show evidence of further growth for at least two weeks. At the end of 7 days young mildew infections were scarcely evident. Although the growth of long-standing infections was arrested, it usually required two or occasionally three sprays at 6- or 7-day intervals to eradicate them.

It was evident from these preliminary tests that Iscothan was superior to both copper sulfate and sulfur in controlling mildew. Its residual protective action was demonstrated in the following test. Ten cucumber plants were sprayed with Karathane, and another ten plants were left unsprayed as a check. Mildew conidia were shaken over the leaves of both sets of plants three hours after the spraying was completed. Whereas no mildew infections developed on the sprayed leaves, numerous infections developed on the unsprayed leaves. In other tests where leaves were exposed to a light daily inoculation new infections did not appear until 14 to 15 days after the fungicide was applied. Since the incubation period of the mildew fungus is 6 or 7 days, it is apparent that the residual protective action of Karathane lasts on sprayed leaves for 7 or 8 days.

Karathane dusts containing 1 to 3 percent active material also afforded excellent control of mildew on greenhouse cucumbers for a period of 6 weeks to 2 months. Eventually, however, mildew spread over the entire lower leaf surfaces, thus showing the inadequacy of the dust treatments. In general, Karathane dust was inferior to the spray in eradicant action.

The effectiveness of Karathane when applied as a smoke[5] was also studied. Three smoke treatments at 6-day intervals in greenhouses where plants supported heavy, long-standing infections eradicated mildew. Although new infections developed subsequently when wind-borne conidia entered through open ventilators, subsequent smoke treatments at fortnightly intervals kept infections at low non-destructive levels. The eradicant action of the smoke was similar, but slightly slower than that of the spray. Nevertheless, the superiority of the smoke treat-

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[3] The microsol dispenser was kindly provided by Mr. L. Spicer, Kemsan Products Limited, Oakville, Ontario.

[4] Dinitro 1-methyl heptyl phenyl crotonate was originally manufactured by Rohm and Haas Company, Philadelphia, as Arathane and later as Karathane. While manufacture of this chemical was discontinued by that company, the material was taken over by Innis, Speiden and Co. Inc., New York, and was sold as Iscothan. More recently Rohm and Haas has resumed the manfacture of Karathane.

[5] Karathane smoke generators and other forms of Karathane were kindly supplied by Mr. Frank Fisher, Plant Products Company, Port Credit, Ontario.

ment over the spray was reflected in the overall increased control.

The residual protective action of the smoke, which was similar to that of the spray, was demonstrated in the following test. Twenty-four hours prior to inoculation with mildew conidia ten cucumber plants were exposed to a Karathane smoke treatment while another ten plants were left unexposed. Average numbers of mildew infections on the inoculated untreated and treated leaves were 87 and 2, respectively. Further tests demonstrated the residual protective effect of the smoke to be of 7 or 8 days' duration under conditions of natural infection.

Although Karathane was observed to be much less phytotoxic than copper sulfate, it occasionally caused injury of a varying degree to the tender cucumber foliage. When it occurred, injury was more severe after spraying than after dust or smoke applications. Severe spray injury consisted of chlorosis and necrosis of marginal and elongate intercostal areas of the leaf laminae. Karathane dusts did not cause more than a mild chlorotic speckling of the youngest leaves. Injury from the Karathane smoke resembled that caused by the spray. In addition, the leaves of a few plants that were enveloped by the smoke as it was released from containers usually became yellowed. This injury was considered to be mainly due to the high temperature of the smoke at the time of emission.

Because of its phytotoxicity certain investigators in England (4) have considered the safety of Karathane as a spray for mildew control to be doubtful. In contrast, numerous tests conducted in commercial greenhouses in southern Ontario have shown that sprays containing minimum effective dosages of Karathane do not cause serious injury.

DISCUSSION

While it is not known what constituted the source of inoculum that gave rise to the infection in greenhouse cucumber crops in December of 1951, and again in 1952 and 1953, several possible explanations might be discussed here. That mildew conidia entered greenhouses from infested wild and cultivated cucurbits growing outdoors in Ontario seems unlikely, because the interval of 3 to 4 weeks between killing frosts and the seeding of the winter crop is considered adequate to eliminate the short-lived conidia as a possible source. Although the fungus may have survived in the perfect stage on old plant debris, thereby giving rise to later infections, the writer is of the opinion that perithecia are an unlikely source since they have never been found on cucumbers in Ontario. Pady's (3) findings of wind-borne spores of several fungi at high altitudes and at great distances from their origin would suggest that mildew conidia from the warmer parts of the United States may be entering southern Ontario late in the year. It also seems probable that powdery mildew may exist in the vegetative stage on another host or hosts in Ontario. In that connection, in January of 1954 the writer found on African violet (Saintpaulia) at Leamington a powdery mildew fungus that infected cucumbers and muskemelons. This isolate grew well on the latter hosts and sporulated abundantly, and it was distinguished from mildew isolates obtained from cucurbits only by its slightly slower growth.

The use of fungicidal smokes in the presence of growing crops is an interesting development in disease control in greenhouse culture. To have a heavily infected crop of cucumbers freed and maintained free from mildew by the regular application of Karathane smokes is a notable advance in the field of chemical control. The general acceptance of Karathane smokes by the grower is due not only to their outstanding effectiveness against powdery mildew but also to the small amount of labor involved in using them. In addition, their appeal to the grower has been enhanced by virtue of the miticidal action of Karathane (2, 5).

The excellent therapeutic effect of Karathane smoke treatments for mildew control is of academic interest as well as of practical importance. The evidence from experiments suggests that control is effected partly by destruction of fungus mycelium that comes into contact with the chemical at the time of its dispersal and partly through the residue left on plant surfaces which prevents conidia that fall upon those surfaces during the following 7 or 8 days from establishing infection.

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RELATIVE RESISTANCE OF STRAWBERRY VARIETIES TO POWDERY MILDEW AT BELTSVILLE, MARYLAND, 1954

George M. Darrow, D. H. Scott, and A. C. Goheen[1]

Summary

Under screenhouse conditions which seemed most favorable to severe powdery mildew infection, 41 strawberry varieties were rated for infection. Eleven varieties were free of mildew and 8 others very resistant. Some others were very susceptible. Under field conditions, slightly less injury was observed than in the screenhouse.

Powdery mildew (Sphaerotheca macularis (Wallr. ex Fries) W. B. Cooke) (= S. humuli (DC.) Burr.) occasionally becomes a major disease of strawberries in the Pacific Coast States and is often serious in England[2]. It is common but less injurious on strawberries in eastern United States. Salmon as early as 1900 reported varietal differences in susceptibility to powdery mildew fungus. For several years varietal differences have also been observed in the strawberry breeding work of the U. S. Department of Agriculture. It is desirable that a classification of mildew resistance be available to strawberry breeders even though this resistance is probably inherited as a multiple factor in strawberries.

At Beltsville it has long been apparent that greenhouse plants in the winter are much more susceptible to powdery mildew than plants of the same variety in the field. This greater susceptibility is probably due in part to the difference in succulence as Tapke[3] showed for powdery mildew of barley and wheat. This same condition holds for plants growing in screenhouses where shade makes plant growth more succulent and the screen lessens air circulation.

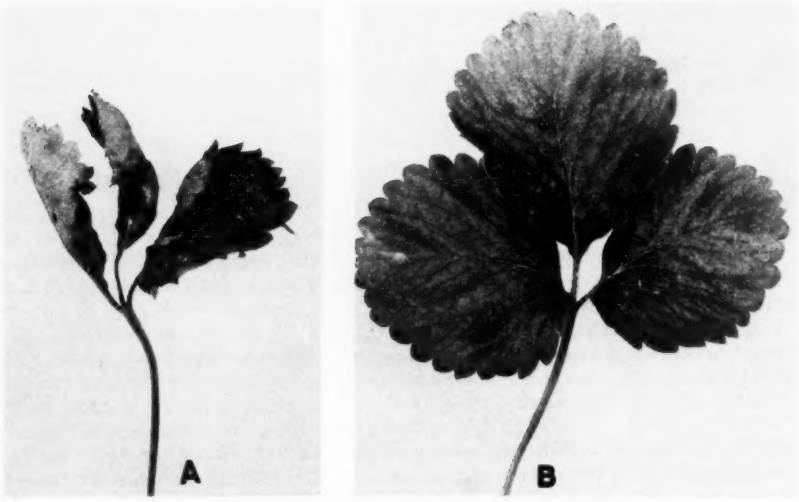

FIGURE 1. A, Armore leaf severely affected with mildew which has distorted the leaflets and caused necrosis. B, Catskill leaf highly resistant to mildew.

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[2] Salmon, E. S. 1900. The strawberry mildew. Jour. Roy. Hort. Soc. 25: 132-138.

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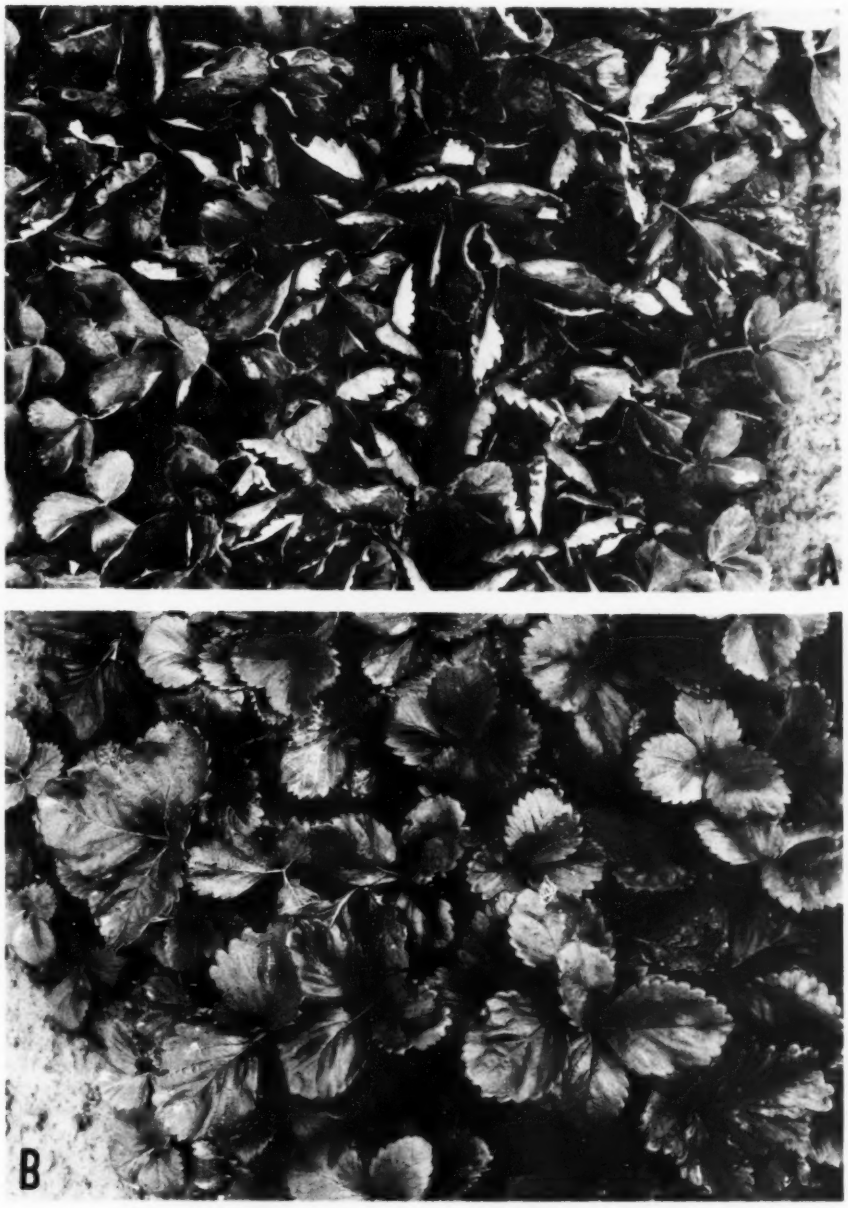

FIGURE 2. A, Robinson strawberry with leaves curled and injured by powdery mildew. B, Sparkle, highly resistant to mildew.

In 1954 virus-free stocks of 41 varieties of strawberries were maintained in a screenhouse at Beltsville where conditions were ideal for epiphytotic development of powdery mildew. This gave an excellent opportunity for comparison of the principal strawberry varieties.

Ratings (Table 1) were made in September 1954, using 10 for freedom from mildew and 1 for severest infection (killing). Three people rated all plots independently and the values presented are averages of the three ratings. Where they were available, the same varieties growing in a nearby field, on low land, were rated. Injury to most varieties consisted in slight leaf

Table 1. Resistance of strawberry varieties to powdery mildew in the field and in the screenhouse, Beltsville, Maryland, 1954[a].

Variety	Rating		Variety	Rating	
	In field	In screenhouse		In field	In screenhouse
Catskill	10*	10	Donner	-	7
Dunlap	10	10	Klonmore	7	7
Empire	-	10	Massey	8*	7
Klondike	10	10	Redstar	8*	7
Konvoy	-	10	Robinson	7*	7
New York	10	10	William Belt	9	7
Orland	-	10	Blakemore	7*	6
Sioux	10	10	Dixieland	7*	6
Sparkle	10*	10	Fairfax	8*	6
Tahoe	-	10	Gem	10	6
Tennessee Shipper	10	10	Howard 17 (Premier)	8*	6
Aroma	9	9	Marion Bell	-	6
Sierra	-	9	Midland	7*	6
Albritton	9	8	Tennessean	-	6
Lassen	-	8	Tennessee Beauty	7*	6
Marshall	-	8	Vermilion	9	6
Missionary	9	8	Stelemaster	6	5
Parramos	10	8	Ambato	-	2
Pocahontas	9*	8	Armore	4*	2
Bellmar	8*	7	Royal Sovereign	-	2
Climax	9	7			

[a] 10 = no mildew and 1 = killing by mildew.
* Average for 5 replicate plots of each.

rolling -- rating 9 and 8; considerable leaf rolling -- rating 7; much leaf rolling and some injury to the undersurface (reddening and beginning of necrosis) -- rating 6. A rating of 5 indicates injury severe enough to cause serious loss of leaves. On the Marshall variety leaf rolling did not occur; instead the leaves were considerably mottled. No other variety had just this type of injury. Ambato, Armore, and Royal Sovereign were the varieties most severely injured by mildew.

The ratings below 5 given to certain varieties are probably not completely accurate. While these varieties may be highly susceptible to mildew, they are also susceptible to parathion injury. Parathion dust was periodically necessary to control insect infestations of the plants. It was not determined that powdery mildew predisposed the plants to greater parathion damage or that a common factor predisposed the plants both to attack by powdery mildew and to injury from parathion.

The number of varieties for each rating in the screenhouse is as follows:

Rating	Varieties with indicated rating (number)
10	11
9	2
8	6
7	8
6	10
5 or below	4

HORTICULTURAL CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, U. S. DEPARTMENT OF AGRICULTURE

EXPERIMENTS ON CONTROL OF BACTERIAL CANKER OF BLUEBERRY

Edward K. Vaughan and C. A. Boller[1]

Bacterial canker[2] frequently causes severe losses in cultivated blueberries in Oregon, particularly on the varieties Jersey, Atlantic, Scammell, Coville, Evelyn, and N51G. The cankers form on one-year-old stems. When a canker girdles a stem, the stem dies above the girdle. When a canker does not girdle a stem, the stem is weakened to an extent depending on the size of the canker. The disease can kill young plants and can destroy over half of the one-

Table 1. Control of bacterial canker of blueberry by spraying with Bordeaux mixture 1952-53.

Treatment	Percentage of plants diseased*	Average number infected stems per plant	Average number cankers per plant
Bordeaux spray	42.6	0.57	0.59
Unsprayed	96.3	5.39	6.33

* 54 plants per treatment.

Table 2. Control of bacterial canker of blueberry by spraying with Bordeaux mixture 1953-54.

Treatment	Percentage of plants diseased*	Average number infected stems per plant	Average number cankers per plant
Bordeaux spray	12.50	0.29	0.33
Unsprayed	58.33	1.71	2.17

* 24 plants per treatment.

Table 3. Control of bacterial canker of blueberry by spraying with various fungicides 1953-54.

Material used	Percentage of plants diseased*	Average number infected stems per plant	Average number cankers per plant
Bordeaux 8-8-100	31.25	.44	.56
Mathieson 466 2 lbs/100 gallons	50.00	1.31	1.71
Ethyl N622 1 1/2 lbs/100 gallons	68.75	2.35	3.42
Copper "A" 6 lbs/100 gallons	25.00	.31	.49
Check, not sprayed	56.25	1.38	2.06
L.S.D. .05	12.44	0.48	--

* 16 plants per treatment.

[1] Respectively Plant Pathologist, and Assistant Horticulturist, Oregon Agricultural Experiment Station.

[2] Stace-Smith, R., P. H. Wooley, and Edward K. Vaughan. 1953. A new disease of cultivated blueberry. Phytopath. 43: 589. (Abst.).

year-old stems on older plants.

In the fall of 1952 the blueberry planting at the Lewis-Brown Horticultural Farm was divided into three blocks of 54 plants each. One block was sprayed with Bordeaux mixture 8-8-100, one with Puratized Agricultural Spray 1 pint per 100 gallons, and the third was not sprayed. The treatments were not replicated because the etiology of the disease had not been definitely established and attempts to control it were on a purely empirical basis.

First applications were made October 9, 1952. Within five days the plot sprayed with Puratized Agricultural Spray was partially defoliated, most of the remaining leaves were severely burned, and there were numerous dark lesions on the stems. No further applications of this fungicide were made. Second application of Bordeaux mixture was on November 6, 1952.

In 1953 the disease appeared very early in the season. The number of infections did not increase between February 28 and March 28. The results showed a substantial reduction in the number of infections in the plants sprayed with Bordeaux mixture (Table 1).

Randomized, replicated plots were sprayed with Bordeaux mixture 8-8-100 on October 7 and November 4, 1953. In 1954 the disease appeared later in the season, and the number of infections was not determined until April 14. Although the disease was not so severe as in 1953, the results (Table 2) were similar and again demonstrated that Bordeaux mixture applied in October and November will significantly reduce the severity of the disease.

In a second test in 1953-54 Bordeaux mixture and three other fungicides were applied to randomized, replicated plots on October 7 and November 4. The results (Table 3) confirm the effectiveness of Bordeaux mixture and indicate that other copper materials can also be used for control of this disease.

In both 1953 and 1954 it was noted that the bases of buds on plants sprayed with copper fungicides turned dark brown or almost black. This discoloration was so consistent that it could be used to determine the distribution of the fungicide. Buds with discolored bases developed normally the following season.

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LATE SEASON PROTECTIVE AND ERADICATIVE SPRAYS AS MEANS OF CONTROLLING BROWN ROT OF PEACH FRUIT

Joseph M. Ogawa, Ross Sanborn, Harley English, and E. E. Wilson[1]

Summary

Spray preparations containing captan, one of the carbamate compounds, or wettable sulfur, appear to afford good protection to peach fruit against infection by the brown-rot fungus. Of the materials tested for eradicative action, lime-sulfur proved effective in arresting the development of infection which occurs during rains. This material, at 2 gallons per 100 gallons of water, applied 38 hours after the beginning of a rain which initiated the infection, prevented further development of the fungus in the fruit. In the event of rain during harvest, therefore, a combination of lime-sulfur and wettable sulfur offers a means of arresting incipient infection and protecting fruit against further infection under California conditions.

During the 1954 harvest season of canning peaches, considerable amounts of lime-sulfur and lime-sulfur plus wettable sulfur were applied by growers after a rain in late August. This practice probably prevented severe loss from brown rot. In some orchards fruit drop was heavy during this period. No conclusive evidence was found that lime-sulfur or other spray materials used in the orchards caused this drop. Excessive phytotoxicity was not observed in any of the sprayed orchards.

Again in 1954, brown rot, incited by Sclerotinia (Monilinia) fructicola, reduced the peach crop in the central California peach districts. This is the third consecutive year in which severe losses have been incurred. In each of the years, a short rain gave impetus to the disease in peach varieties that were nearly ripe or ripe at that time. In the epidemic of 1948, however, exceptionally heavy dews at night probably contributed to the severity of infection.

With this relation of moisture to infection in mind, the experimental work in 1954 consisted of both pre-infection and post-infection treatments of fruits with various fungicides.

Materials and Methods

Trees grown under commercial orchard conditions in Butte and Sutter Counties were used in the experiment. Each spray treatment was given to 10 to 15 fully mature trees. The varieties used, in the order in which they were tested and in the order of their maturity, were Fortuna, Peak, and Sims. During 1954 sprays were applied three to four weeks and one week before harvest maturity unless otherwise indicated. To increase disease development the trees were sprayed with water during a night within three to six days after the second spray application. Data as to the percentage of diseased fruits were taken in the orchard five to six days after the simulated rain.

The trade and common or chemical names of the spray materials used were as follows: Orthocide 50W (captan), Zerlate (ziram), Mathieson Bulb Fungicide No. 275 (pentachloronitrobenzene), Flotox Wettable Sulfur (wettable sulfur), Purex (sodium hypochlorite), Phygon XL (dichlone), Manzate (maneb), Vancide 51ZW (zinc dimethyl dithiocarbamate; zinc 2-mercaptobenzothiazole), lime-sulfur (calcium polysulfide), Dowicide A (M-244) (purified sodium orthophenylphenate), Isothan Q15 (lauryl isoquinolinium bromide), Polysulfide Compound (sodium polysulfide), XSF#3 (H. L. Woudhuysen and Associates, experimental silver fungicide), Tag 331 (phenylmercuric acetate), and Puratized Apple Spray (phenyl mercuric monoethanol ammonium acetate).

Protective Measures

Experimental data for 1953 are shown in Table 1. The data show that Orthocide 50W consistently afforded good protection in three separate trials. Zerlate and Mathieson Bulb Fungicide No. 275 also appeared promising. Two applications of either Orthocide 50W or Purex appeared to be more effective than one.

In a preliminary trial during 1954, eight spray materials were applied on Fortuna peach

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[1] The authors wish to express their appreciation to Fred Peterson and Walter M. Anderson, farm advisors of Sutter and Yuba Counties respectively, for their assistance in this investigation.

Table 1. The use of preharvest fungicidal sprays for the control of brown rot in peaches. 1953.

Chemical	Material per 100 gals. spray	No. of sprays[a]	Average percent diseased fruits		
			Stuart[b]	Walton[c]	Johnson[c]
Orthocide 50W	2.0 lbs.	1	28.2	50.3	16.7
Orthocide 50W	2.0 lbs.	2	--	12.7	12.7
Zerlate	2.0 lbs.[d]	1	39.2	--	--
Mathieson Bulb Fungicide No. 275	6.0 lbs.	1	52.7	--	--
Flotox Wettable Sulfur	6.0 lbs.	1	54.7	67.0	--
Purex	1 gal.	1	61.0	91.7	87.3
Purex	1 gal.	2	--	63.0	33.7
Check	--	--	81.2	96.0	91.7
L.S.D. 5%			24.7	44.3	22.7
L.S.D. 1%			33.3	63.1	33.1

[a] (1) 6 days before harvest. (2) 6 and 11 days before harvest.
[b] Fruits dusted with spores and kept wet for 7 1/2 hours during the night to induce disease.
[c] Fruits not dusted with spores. Fruits picked at random from plots and incubated in a moist chamber to determine the effectiveness of control.
[d] Plus 2 oz. duPont Spreader-Sticker.

Table 2. The use of preharvest fungicidal sprays for the control of brown rot in peaches. 1954.

Chemical	Material per 100 gals. spray	Fortuna		Peak	
		% diseased fruits	Spray injury[c]	% diseased fruits	Spray injury
Flotox Wettable Sulfur	10 lbs.	4.6	0	3.9	0
Phygon XL	3/4 lb.	3.9	+++	---	---
Phygon XL	1/2 lb.	---	---	4.4	+++
Orthocide 50W	2 lbs.	3.1	0	4.6	+
Manzate	2 lbs.[a]	3.0	0	4.0	++
Zerlate	2 lbs.[a]	6.6	0	---	---
Vancide 51ZW	2 lbs.[b]	9.5	0	---	---
Mathieson Bulb Fungicide No. 275	6 lbs.	8.2	0	---	---
Purex	1 gal.	6.3	0	---	---
Check	---	11.6	0	17.6	00
L.S.D. 5%		2.5		4.4	
L.S.D. 1%		3.6		6.1	

[a] Plus 1 oz. duPont Spreader-Sticker.
[b] Plus 1 oz. Triton X-100.
[c] 0 None, + Slight, only skin deep, ++ Moderate, only skin deep, +++ Severe, into flesh.

trees. In an additional test four of the most promising materials were applied to the Peak variety. The results of both trials are presented in Table 2.

On Fortuna, all treatments were significantly better than the check at the 1% level, except Mathieson Bulb Fungicide No. 275, which was significantly better than the check at the 5% level and Vancide 51ZW which was not significantly better than the check. There were no significant differences in the results obtained with Orthocide 50W, Phygon XL, Flotox Wettable Sulfur, and Manzate. Orthocide 50W and Manzate were significantly better than Zerlate at the 5% level and better than Vancide 51ZW and Mathieson Bulb Fungicide No. 275 at the 1% level. In the trials on Peak with the four most promising compounds, all of the treatments reduced the percentage of diseased fruits significantly (1% level) as compared with the check. Similar results were obtained with all of the materials.

Phygon XL at 3/4 and 1/2 lb. per 100 gallons of spray produced injury to the fruits which extended into the flesh. Careful observations were made to determine the extent of the injury in two orchards where the material was applied with a speed sprayer. The surface of the fruit facing the sprayer and those fruits located closest to the spray nozzle had the greatest injury (distinct lesions), whereas least damage occurred to peaches high in the tree. Fruits in one orchard were more severely injured than those in the other, even though the spray treatments were said to be the same. These facts appear to indicate a correlation between spray coverage and injury. It is suggested that Phygon XL might serve as a good material to determine spray coverage on peach fruits with various types of spray equipment.

Trees of the Sims variety were sprayed with spores, and then sprayed with water in an attempt to obtain a high incidence of disease. In addition to artificial moisture the trees were wetted by rain.

The data in Table 3 show that all treatments were significantly better than the check at the 1% level, whereas no significant differences existed between treatments.

Eradicative Sprays

Because the occurrence of the brown-rot disease is erratic, many of the orchardists do not apply protective measures. Precipitation just before or during the haryest season in these orchards frequently results in heavy losses, and even in orchards receiving protective sprays, serious amounts of decay sometimes occur. In the absence of rain, brown-rot epidemics in California are infrequent. In view of these facts, it would be highly desirable to have a spray material which would act as an eradicant of incipient fruit infections.

Loss of fruit is negligible if fruits are picked and canned within 72 hours after beginning of rain. It is on the fruits that cannot be picked and canned within a short time after a rain that an eradicative treatment would be valuable.

Control of brown rot through eradication has been attempted with sodium hypochlorite by applying the spray after the disease was showing[2]. Fairly effective control was reported. During 1953, immediately after a rain, sodium hypochlorite was again applied but the results were inconclusive[3].

It would appear that the most effective timing for eradicative sprays would be immediately after precipitation.

Preliminary laboratory tests were designed to determine the eradicatory properties of the following materials: Purex, XSF No. 3, Orthocide 50W, Manzate, Mathieson Bulb Fungicide No. 275, Flotox Wettable Sulfur, Phygon XL, Isothan Q15, Dowicide A (M-244), and lime-sulfur. In addition, Tag and Puratized Apple Spray, materials known to possess eradicatory properties, were tested, though they could not be used commercially because of their mercury content. Promising results were obtained with Tag, Puratized Apple Spray, and lime-sulfur. These materials were then used in orchard tests, the results of which are shown in Table 4.

The data in Table 4 show that lime-sulfur, Purex, and Isothan Q15 were the most effective as eradicant fungicides.

Lime-sulfur and Polysulfide Compound were then tested on Sims peaches for eradicative action. First the trees were sprayed with a spore suspension, and later, the same evening, moisture was applied with an air-blast sprayer so as to keep the trees continually wet for an

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[2] Sanborn, Ross. 1953. Gridley grower finds household bleaching agent effective for control of brown rot on cling peaches. Western Fruit Grower 7 (3): 36.

[3] Sanborn, Ross A. 1953. Peach growers test brown rot control. Western Fruit Grower 7 (11): 21-22.

Table 3. The use of preharvest fungicidal sprays for the control of brown rot in Sims variety of peach. 1954.

Chemical	Lbs. per 100 gals. spray	Percent diseased fruit	
		Two sprays	One spray[b]
Orthocide 50W	2	13.7	5.5
Manzate[a]	2	16.4	10.6
Flotox Wettable Sulfur	10	19.1	14.9
Zerlate[a]	2	21.5	---
Check	---	43.6	39.8
L.S.D. 5%		9.7	11.2
L.S.D. 1%		13.3	15.5

[a] Two oz. of duPont Spreader-Sticker added per 100 gallons of water.

[b] Applied 1 week before the addition of moisture.

Table 4. The effectiveness of fungicides applied as eradicants for the control of brown rot in Peak peaches, Butte County, 1954[a].

Chemical	Amount per 100 gals. spray	Percent diseased fruits	Spray injury
Lime-sulfur	2 gals.	16.2	++[b]
Lime-sulfur	0.5 gal.	27.0	+
Purex	4 gals.	26.7	0
Manzate	4 gal	35.2	+++
Dowcide A (M-244)	4 pts.	35.4	+
Orthocide 50W	4 lbs.	38.7	+
Isothan Q15	2 pts.	32.6	+
Check	--	52.6	0
L.S.D. 5%		17.7	
L.S.D. 1%		23.8	

[a] The trees were sprayed with a spore suspension, and moisture was maintained on the fruits for 13 hours before eradicants were applied.

[b] 0 None, + Very slight, ++ Slight, +++ Moderate.

[c] Plus 2 oz. of duPont Spreader-Sticker.

Table 5. Polysulfides as eradicant sprays for the control of brown rot in Sims peaches. Butte County, 1954.

Chemical	Amount per 100 gals.	Percent diseased fruits
Lime-sulfur	3 gals.	2.5
Lime-sulfur	2 gals.	3.0
Lime-sulfur and Flotox Wettable Sulfur	10 lbs.	2.4
Polysulfide Compound	2.4 lbs.	4.7
Check		29.3
L.S.D. 5%		8.1
L.S.D. 1%		11.1

infection period of 13 hours. Immediately after the infection period, various spray concentrations of polysulfides were applied at the rate of 5 to 6 gallons per tree. The results are given in Table 5.

The data in Table 5 indicate that these polysulfide materials were effective in the eradication of incipient infections when applied 13 hours from the beginning of moisture application. No significant differences were found between the various treatments. The mixture of liquid-lime sulfur and wettable sulfur appeared to give results as satisfactory as lime-sulfur alone.

Further experimentation was designed to determine how soon eradicants should be applied after a rain. An application of lime-sulfur, 2 gallons per 100 gallons of water, 38 hours after the beginning of precipitation appeared to give as good control as applications made 14, 17, and 20 hours after the beginning of precipitation. These tests were made on a small scale, so further trials are necessary before specific recommendations can be made.

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FIREBLIGHT CONTROL WITH SPRAYS OF AGRI-MYCIN, A STREPTOMYCIN-TERRAMYCIN COMBINATION[1, 2]

Robert N. Goodman[3]

The successful control of fireblight (Erwinia amylovora) of apples with antibiotics in orchard and greenhouse experiments in 1952 and 1953 (3, 4) prompted further investigations in 1954.

Results from the 1953 orchard experiments demonstrated that combinations of streptomycin and Terramycin[4] and streptomycin alone, applied as foliar sprays, at concentrations ranging from 500-100 ppm, provided complete control of this most serious bacterial disease of apples. Perfect blight control was obtained where seven and six antibiotic sprays were applied at four-day intervals beginning at full pink and 30 to 50 percent of full bloom respectively. Schedules of five and four sprays, initiated post-bloom at early petal fall and calyx also provided excellent control; however, some cluster and shoot blight was evident. Data from the 1953 experiments strongly suggested that the antibiotic sprays had acted as both systemic protectants and systemic eradicants.

Effective control of fireblight of apples and pears with antibiotic sprays has also been reported by Ark (1), Heuberger and Poulos (5), Winter and Young (6) and most recently by Dunegan (2).

The 1954 Orchard Experiments

The 1954 experiments were conducted in nine commercial orchards strategically located throughout the apple producing areas of Missouri. With the exception of the three test plots at Boonville, Missouri, all sprays were applied with speed sprayers. At Boonville, the sprays were applied from the ground with a single-nozzle hand-gun, delivering approximately 10 gallons per minute at 600 pounds pressure psi.

All plots, with the exception of plots 2 and 3 at Boonville, consisted of either two or three adjacent tree-rows and these were subdivided into five to seven tree-blocks. Control trees were located at the row-ends in each plot. At Boonville, plots 2 and 3 consisted of several adjacent rows wherein antibiotic-sprayed trees were alternated with control trees. The controls in all plots were sprayed four times with water.

Fireblight infections were counted four times at approximately ten-day intervals. The final counts were made on June 22-24. By June 22 fireblight had presumably run its course since new infections were no longer discernible.

As in 1953, blossom cluster counts and subsequently counts of fruit set were made in several test plots to determine the effect that in-bloom and post-bloom antibiotic sprays had on fruit set and development. In addition, samples of fruit from antibiotic-treated and control trees were obtained at harvest to observe any impairment of fruit finish that might be attributed to the antibiotic spray material.

The trees utilized in these experiments were exclusively of the extremely blight-susceptible Jonathan variety.

Results

Protective-Type Antibiotic Sprays: Fireblight was present to an appreciable degree in five of the nine orchards where sprays were applied. Results from the protective-type antibiotic sprays are presented in Table 1. There was considerable variation in the severity of fire-blight from orchard to orchard and occasionally between plots within an orchard. For this reason effectiveness of the antibiotic spray treatments can best be evaluated by comparing antibiotic-treated trees with controls of the same plot. The results may be interpreted as follows:

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[1] Journal Series Paper No. 1489 approved by the Director of the Missouri Agricultural Experiment Station.

[2] Agri-mycin, a trademarked formulation of Chas. Pfizer and Company, Inc., Brooklyn, New York, which contains 15 percent streptomycin and 1 1/2 percent Terramycin.

[3] Assistant Professor of Horticulture, University of Missouri, Columbia, Missouri.

[4] Terramycin is the trade name of Charles Pfizer & Company, Inc., for oxytetracycline.

Table 1. Effectiveness of Agrimycin protective-type antibiotic spray programs for fire-blight control-1954.

Location[a]	Number sprays	Agrimycin conc. (ppm)[b]	Number trees	Blight, number per tree		Spray dates[e]
				Clusters[c]	Shoots[d]	
West Seymour	4	100	20	0.2	1.3	April 12, 16, 20, 24
	3	100	20	0.25	1.25	April 12, 16, 20
	2	100	20	2.0	8.0	April 12, 16
	4	control (water)	20	7.0	59.0	April 12, 16, 20, 24
East Seymour	4	100	20	1.3	2.5	April 12, 16, 20, 24
	3	100	20	1.5	3.0	April 12, 16, 20
	2	100	20	4.0	13.1	April 12, 16
	4	control (water)	20	18.0	71.0	April 12, 16, 20, 24
Boonville (1)	3	100	24	2.2	4.5	April 20, 28, 28
	4	control (water)	19	28.0	54.0	April 16, 20, 24, 28
Boonville (2)	4	50	13	4.0	14.6	April 16, 20, 24, 28
	3	50	12	6.6	16.6	April 16, 20, 24
	4	control (water)	23	50.0	98.0	April 16, 20, 24, 28
Boonville (3)	4	25	13	0.9	4.8	April 16, 20, 24, 28
	3	25	11	2.8	12.5	April 16, 20, 24
	4	control (water)	24	12.0	30.0	April 16, 20, 24, 28
Waverly	3	100	10	1.1	11.7	April 19, 23, 27, May 3
	3	100	10	4.7	9.1	April 19, 23, 27
	2	100	10	10.0	42.5	April 19, 23
	4	control (water)	12	50.0	69.0	April 19, 23, 27, May 3
Savannah	4	100	7	5.0	41.0	April 25, 29, May 3, 8
	3	100	7	2.0	30.0	April 25, 29, May 3
	4	50	14	4.0	55.0	April 25, 29, May 3, 8
	3	50	14	4.0	28.0	April 25, 29, May 3
	4	control (water)	21	55.0	244.0	April 25, 29, May 3, 8

[a] Age of trees: at West Seymour, 20 years; East Seymour, 25 years; Boonville 12 years; Waverly 12 years; Savannah 14 years.

[b] Concentration of Agrimycin-100, powdered form, with the exception of Savannah plot where Agrimycin-200, a liquid concentrate containing 35% Streptomycin, was used.

[c] Clusters blighted per tree (primary infection).

[d] Shoots blighted per tree (secondary infection).

[e] Spray dates: All spray schedules initiated at approximately 5 to 10 percent of full bloom with the exception of Boonville (1) plot, where first spray was applied 2 days after full bloom.

Table 2. Influence of spray-plot design on secondary fireblight infection -- Savannah, Missouri 1954.

Tree No.	No. of sprays	100 ppm.[a] Observation date: June 9 cb	June 9 sb	June 24 cb	June 24 sb	50 ppm.[a] Observation date: June 9 cb	June 9 sb	June 24 cb	June 24 sb	50 ppm.[a] Observation date: June 9 cb	June 9 sb	June 24 cb	June 24 sb
1	3	0	0	0	16	2	5	3	36	2	5	3	31
2	3	2	2	2	58	19	33	20	77	2	7	3	56
3	3	6	12	6	46	9	14	9	50	1	3	4	7
4	3	2	4	3	31	1	4	1	13	3	6	3	12
5	3	0	0	0	12	4	11	4	57	0	0	0	4
6	3	2	6	2	37	1	2	1	13	0	0	0	5
7	3	0	1	0	7	0	0	0	9	1	1	1	17
8	4	0	2	1	43	0	0	1	29	3	4	3	29
9	4	12	35	12	44	2	12	4	33	0	0	0	3
10	4	1	0	3	28	0	1	1	21	1	0	1	8
11	4	0	0	0	1	1	7	2	23	2	0	2	13
12	4	4	0	4	9	2	5	2	23	2	8	5	97
13	4	3	0	3	11	6	20	10	156	11	71	13	134
14	4	8	18	14	150	4	24	5	96	8	17	11	103
15	B[b]	28	52	28	96	18	70	23	241	23	38	24	124
16	C[c]	34	66	41	231	126	250	150	310	73	142	79	244
17	C	48	161	56	209	42	113	61	270	51	120	63	328
18	C	23	39	31	91	37	128	49	351	43	131	49	211
19	C	48	112	53	204	21	33	24	134	26	69	30	245
20	C	31	52	40	127	23	54	30	222	31	77	35	226
21	C	110	318	120	380	27	143	33	313	21	48	23	171
22	C	76	191	110	342	41	137	51	401	24	37	35	129

[a] cb and sb = cluster blight and shoot blight respectively.

[b] B = Buffer, sprayed 4 times with antibiotics but regarded in tabulation of data as neither a treated nor a control tree.

[c] C = Control tree, these trees were sprayed 4 times with water.

1. Antibiotic sprays, regardless of number or concentration, very markedly reduced the number of cluster and shoot blight infections.
2. There was little difference in the degree of blight control provided between the four- and three-spray treatments at either 100 or 50 ppm.
3. The four-spray schedule at 25 ppm appeared to be equally as effective as either four or three sprays at the higher concentration.
4. The difference in effectiveness between the four- and three-spray program at 25 ppm may be more apparent than real. One tree in the 11 receiving three sprays at 25 ppm had 70 percent of both the cluster and shoot blight infections recorded for this block.
5. In all instances the two-spray schedule was less effective than the three- or four-spray program. The degree to which this treatment was inferior varied directly with the severity of blight within the block.
6. The single instance of a spray program initiated after full bloom, the Boonville (1) plot, furnished excellent control of primary and secondary blight.

From the foregoing data it is readily apparent that fireblight was appreciably reduced by antibiotic sprays regardless of the concentration employed or the number of sprays applied. However, it is also evident that primary and secondary infection were present in all antibiotic-treated blocks. In some blocks secondary infection occurred to an appreciable extent and the amount appeared to vary directly with; (i) the proximity of the antibiotic-treated trees to the water-sprayed controls and (ii) the intensity of the fireblight attack in the orchard. The data in Table 2 clearly illustrate the effect that water-sprayed controls had on antibiotic-treated trees. Spread of blight from the controls to the four-spray treatment caused the three-spray schedule to appear superior.

Table 2 also shows that there was an extensive delay in the appearance of significant amounts of secondary infection in the antibiotic-treated blocks. Blight control was excellent on June 9; however, by June 24 considerable secondary infection had occurred. Primary blight was first observed at Savannah on May 13 and the last antibiotic spray was applied on May 8.

The profound influence of spray-plot design on distribution of secondary infection and the delayed appearance of appreciable amounts of shoot blight were not unique to the Savannah plots. Similar conditions prevailed at Boonville in plots 2 and 3, where control trees were alternated with antibiotic-treated trees, and at Waverly.

Comparative Severity of Secondary Infection in 1953 and 1954: It was observed that secondary infection was decidedly more severe, where fireblight was a problem, in 1954 than it was in 1953. It is suggested that the difference in rainfall between the two years is a contributory factor. The rainfall data for Boonville given in Table 3 are indicative of precipitation not only at Boonville (central Missouri) but also at Waverly and Savannah (north-central and northwestern Missouri). In southwestern Missouri in the Seymour area precipitation was unusually light both in 1953 and 1954.

Table 3. Rainfall at Boonville, Missouri

	1953	1954
May 1-31	2.34 in.	5.29
June 1- 7	0.52	2.01

Post-Infection Eradicative Sprays: The efficacy of Agrimycin, applied as an eradicant, was evaluated in three orchards. Two sprays containing the antibiotic combination at 100 ppm were applied at three-day intervals after blight symptoms had become evident.

The time lapse between the observance of primary infection and the application of the first of two antibiotic sprays in the three orchards varied from two to five days. The results from the eradicative-type sprays in the three test orchards were approximately the same and only the data from the Waverly test plot appears in Table 4.

In each instance the antibiotic sprayed trees had, at the conclusion of the experiment, fewer infections than the water-sprayed controls. However, eradication was not complete and the sprays seemed merely to delay temporarily the further spread of infection.

Phytotoxic Effects of Antibiotic Sprays: Two years' data concerning the effect of antibiotic sprays on fruit set, development, finish, and foliage injury may be summarized as follows:

1. As many as six in-bloom and post-bloom sprays at concentrations up to 100 ppm streptomycin did not adversely affect fruit set, development, or finish.
2. Slight marginal chlorosis was observed on some foliage receiving four antibiotic sprays at 100 ppm. A lesser amount was visible at 50 ppm and none was noticeable at the 25 ppm concentration.

Summary

There appeared to be little difference in the effectiveness of blight control between the four- and three-spray program at either 50 or 100 ppm of the antibiotic combination. In addition, a four-spray schedule at 25 ppm also provided a comparable high degree of control. The two-spray program was not particularly effective where secondary infection was a problem.

Although secondary blight was severe in several plots, trees receiving four or three

sprays, at 100 or 50 ppm, remained relatively free of these late infections for as long as 30 days after the first primary infections were observed.

Antibiotic-treated trees located adjacent or in close proximity to the water-sprayed controls became severely infested with blight.

An increase in severity of secondary infection observed in 1954 over that in 1953 was believed to be due, at least in part, to the heavier precipitation during May and the first week of June 1954. More than two-and-a-half times as much rain fell during this critical period in 1954 than in 1953.

Eradicative antibiotic sprays applied two to five days after primary blight was observed failed to provide adequate control. At best, this treatment served only to delay the spread of secondary infection.

Fruit set, development, and finish were not impaired by as many as six antibiotic sprays at concentrations as high as 100 ppm. Slight marginal chlorosis of foliage, which has been established as being streptomycin-induced, was occasionally observed where antibiotic sprays were applied at 100 and 50 ppm. At the 25 ppm level this chlorosis was not evident.

Table 4. Eradicative spray block, Agrimycin 100 ppm. Waverly, Missouri -- 1954.[a]

No. of trees	Blight per tree May 5	Treatment	Blight per tree June 24	Blight increase
15	47	2 Antibiotic sprays (100 p.p.m.)	320	6.8 times
17	26	2 Water sprays (control)	278	10.7 times

[a] Spray applications: May 5 and May 8. (Blight first observed on April 30).

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UNIVERSITY OF MISSOURI, COLUMBIA

NOTE ON THE INHIBITORY ACTIVITY OF SOME MICROORGANISMS AGAINST PHYTOPHTHORA CINNAMOMI

Mihailo Krstić[1]

Phytophthora cinnamomi Rands is a widespread and important parasite that attacks the roots of seedlings and mature forest trees of numerous species. No effective control has yet been found.

Little attention has so far been given to the possibility of biological control. One experiment, reported by Crandall, Gravatt, and Ryan (1), gave negative results. In their trials decay was not reduced when Trichloderma viride Pers., which is apparently antangonistic to P. cinnamomi, was incorporated into the soil.

In the limited small scale study herein reported, made at the United States Department of Agriculture Plant Industry Station, Beltsville, Maryland, Bacillus subtilis Cohn emend. Prazmowski (strain A 2 of the Forest Research Institute, Belgrade, Yugoslavia), an undetermined species of Penicillium that produces red pigmentation in media, and Pleurotus griseus Pk., all previously reported to possess antagonistic properties for various organisms (2, 3), were tested in culture for possible reduction of growth of P. cinnamomi.

In preliminary tests Petri dishes containing potato-dextrose agar, each seeded with one of these organisms, were inoculated with P. cinnamomi. The distance across which the other organism stopped growth of the Phytophthora was measured in each dish. The inhibitory zones were 6 to 8 mm across with Bacillus subtilis and Penicillium sp., and 12 to 14 mm with Pleurotus griseus (Figs. 1, 2, 3). Some filaments of Phytophthora had grown secondarily into the inhibition zones in the bacterial cultures. In the Phytophthora-Pleurotus and Phytophthora-Penicillium cultures, however, these zones remained definitely clear, showing sensitivity of P. cinnamomi toward these two organisms.

Further tests were conducted to determine if these three organisms produce inhibitory substances for the Phytophthora in broth culture.

Methods

Bacillus subtilis was grown in 20 ml of potato-sucrose liquid (8 gms potato, 2 gms sucrose, to 100 ml distilled water) in 100-ml flasks, at 25° and 34° C in total darkness; and at 22° under continuous fluorescent white light, continuous fluorescent white light filtered through two layers of red cellophane, and in darkness[2, 3]. The Penicillium and Pleurotus griseus were grown in the same medium, but only at 22°, under filtered and unfiltered light and in darkness. After 14 and 21 days the cultures were filtered through a Seitz filter and then added to 5 ml of the original broth at ratios of 1:50, 1:20, 1:10, 1:5, 1:2, and 1:1. The dilutions so prepared were inoculated with mycelium of Phytophthora cinnamomi from 7-day-old cultures and incubated at 25°, in the dark. Readings were taken at 3-, 7-, and 14-day intervals.

Results

Bacillus subtilis

Different Temperatures: Of the filtrates prepared from cultures of B. subtilis grown at 25° C, the 1:20 dilution of the 14-day-old culture and the 1:5 dilution of the 21-day culture retarded growth of Phytophthora cinnamomi at the first reading. No growth appeared in the 1:2 or the 1:1 dilutions from either 14- or 21-day-old cultures. After 7 days filtrates from cultures of both ages produced approximately identical growth inhibition. A very dense and retarded growth of Phytophthora appeared in the 1:2 and 1:1 dilutions, the mycelium growing up from the bottom and occupying 1/3 of the medium. Controls were completely filled up with the

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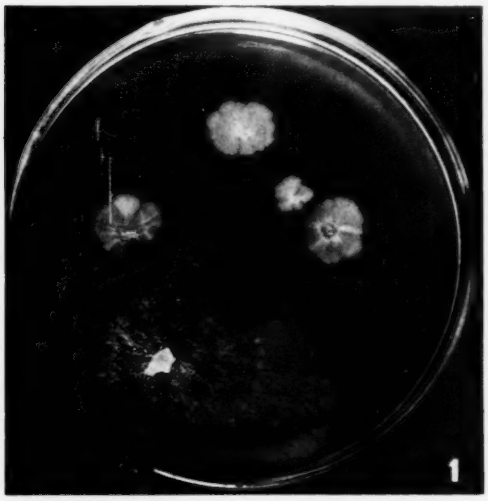
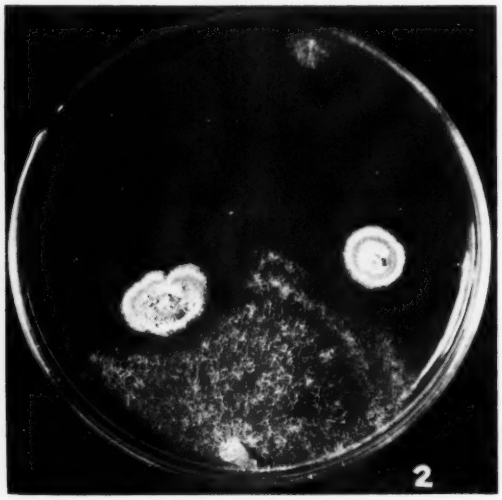
[1] Forest Pathologist, Forest Research Institute, Belgrade, Yugoslavia. On a United Nations fellowship in the U. S. A.

[2] Study of light influences was suggested by G. F. Gravatt, Plant Industry Station, Beltsville, Maryland.

[3] A special installation at the Plant Industry Station, for the study of the physiological effect of light on plants, with uniform and constant temperature (22° C), was provided by Harry A. Borthwick and Wilbur D. McClellan.





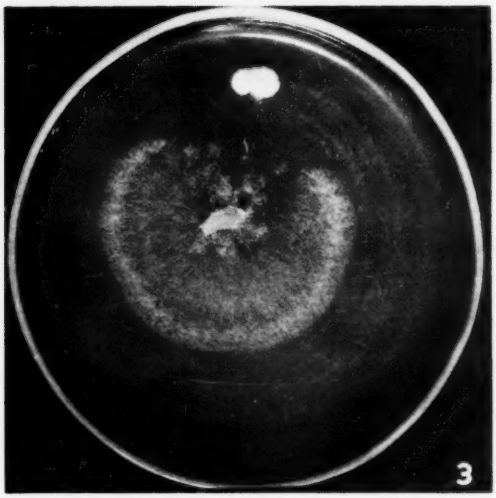


FIGURE 1. Growth of Phytophthora cinnamomi inhibited by Bacillus subtilis. Culture of Phytophthora 6 days old; Bacillus 4 days old. Note secondary growth of mycelium of P. cinnamomi in the inhibited zone, as well as inhibition of growth of the bacterium.

FIGURE 2. Phytophthora cinnamomi inhibited by Penicillium sp. Culture of Phytophthora is 7 days old; Penicillium sp. is 6 days old.

FIGURE 3. A pronounced inhibition of growth of Phytophthora cinnamomi by Pleurotus griseus. Culture of Pleurotus is 7 days old; Phytophthora is 4 days old.

Phytophthora in the same period.

Less inhibition was evident at 3 days in the filtrates from cultures grown at 34° C. At 7 days, however, the inhibitory action of filtrates from cultures grown at both temperatures was identical.

At the end of the 14th day the Phytophthora definitely stopped growing in the 1:2 and 1:1 dilutions of filtrates from both 14- and 21-day old cultures grown at 34° C. In all other cases growth was normal.

Different Light Conditions: No noticeable differences were recorded among effects of filtrates from cultures grown under unfiltered white light, filtered red light, and in darkness. In all cases, at 3 days and 7 days growth of the Phytophthora was halted in the 1:1 dilutions and considerably retarded in the 1:2 dilutions.

After 14 days complete inhibition was evident in the 1:1 dilutions and pronounced retardation in the 1:2 dilutions of filtrates from 21-day-old cultures grown under all three light conditions. With filtrates from the 14-day-old cultures no complete inhibition of Phytophthora was recorded, the mycelium growing throughout the medium.

Penicillium sp.

The mycelium of Phytophthora cinnamomi grew uniformly after 3 days in dilutions of filtrates from 14-day-old Penicillium cultures that had been maintained under fluorescent light, red light, and in the dark. However, mycelial growth was markedly retarded. No growth of Phytophthora occurred in the 1:2 or 1:1 dilutions of filtrates from the cultures grown in the red light or in darkness. After 7 days and 14 days growth was inhibited only in the 1:1 and 1:2 dilutions of filtrates from cultures grown in the dark. All other dilutions and the controls were completely saturated with Phytophthora.

Very pronounced differences were noticed for the 21-day-old cultures of Penicillium. In the first 3 days Phytophthora did not grow in the 1:2 or 1:1 dilutions (continuous fluorescent white light and red light), nor in the 1:10-1:1 dilutions (dark). At the 7-day examination complete inhibition was recorded for dilutions of 1:1 (white light), 1:2 and 1:1 (red light), and 1:5-1:1 (dark). The final examination at 14 days gave the same results.

Pleurotus griseus

Because of the very slow growth of this fungus only 21-day-old cultures were tested. Media for antibiotic production were inoculated with equal-sized particles of agar cultures of Pleurotus, 15 days old.

No inhibitory effect was recorded. In all dilutions the mycelium of the Phytophthora grew naturally, as in the control cultures, and in 7 days filled up the liquid in the test tubes.

Summary

Limited preliminary studies have indicated sensitivity of Phytophthora cinnamomi toward Bacillus subtilis (strain A 2), a species of Penicillium, and Pleurotus griseus, on potato dextrose agar in Petri dishes.

In liquid potato-sucrose medium Bacillus subtilis produced more active inhibitory filtrates at 34° than at 25° C. Definite arresting of growth of P. cinnamomi was noticed after 14 days in dilutions at 1:2 and 1:1 of filtrates from bacterial cultures grown at 34°.

Differential influence of light conditions on inhibitory activity was noted only with the Penicillium. Total inhibition of Phytophthora growth was evident in 1:5-1:1 dilutions of filtrates from Penicillium cultures grown in the dark, 1:2-1:1 from cultures grown under continuous fluorescent white light filtered through red, and 1:1 from cultures grown under continuous fluorescent white light. The reduction of the inhibitory activity of the filtrates from cultures grown under these conditions could be ascribed either to destruction or non-elaboration of antibiotic substances.

These trials were much too restricted to permit definite conclusions. However, the results do suggest the desirability of testing the effects of Bacillus subtilis and the Penicillium sp. on Phytophthora cinnamomi under natural conditions.

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REACTION OF SPRING WHEAT VARIETIES AND SELECTIONS TO ARTIFICIAL INOCULATION WITH LOOSE SMUT[1]

Harry W. Schroeder

Spores of Ustilago tritici, collected from spring and winter wheats in a number of experimental plots and farmers' fields near St. Paul, Minnesota, in 1953, and presumed to contain races of the fungus representative of the area, were used to inoculate plants of 109 varieties and selected lines of wheat grown in yield trials in 1953. Ten heads in each row were inoculated, using the compressed air jet method described by Moore and Munnecke[2]. The seeds from these inoculated plants were planted in head rows in the spring of 1954, and data on loose smut infection were recorded when the plants were fully headed. These data are summarized in Table 1; each figure in the table is an average of the percentage of smutted heads from approximately 10 head rows. The varieties Lee and Mida are the known susceptible checks, and the variety Thatcher is the resistant check. The percentage of smutted heads ranged from 0 to 91; the results suggest that lines of wheat resistant to loose smut can be obtained through adequate and proper selection.

Table 1. Reaction of varieties and selections of spring wheat to Ustilago tritici at St. Paul, Minn. in the 1953-54 season.

No.	Variety or cross	Source	C.I. no. or nursery stock no.	Percent smutted heads
1.	Henry x Mida	Minn.	II-44-71	91
2.	Timstein x Kenya C9906 702-3y-2y-1c-1y-1c	Mexico		88
3.	Egypt 101 x Timstein 704-1y-5y-5c-2c-1c-	Mexico		86
4.	Timstein x Henry	Minn.	C.I. 13026	86
5.	Timstein	Australia	C.I. 12347	80
6.	Y-Kt 2264-1c-4y-3c-1y	Mexico		76
7.	Y (T-K) 2245-1y-1c	Mexico		76
8.	Timstein x Kenya (Blanco)	Mexico		76
9.	Lee	Minn.	C.I. 12488	75
10.	Lerma	Mexico	III-52-1	72
11.	Kenya-Gular-Pilot X K-58-Newthatch	Minn.	C.I. 13047	72
12.	K58 x Mida-Newthatch	Minn.	C.I. 13045	71
13.	Lerma 52	Mexico		70
14.	Ke-Y 2589-9y-6c	Mexico		67

[1] Paper No. 3227, Scientific Journal Series, Minnesota Agricultural Experiment Station, St. Paul 1, Minnesota.

[2] Moore, M. B. and Donald E. Munnecke. 1954. An air blast method for rapid inoculation of wheat and barley with loose smut. Phytopath. 44: 499. Abs.

Table 1. (continued).

No.	Variety or cross	Source	C.I. no. or nursery stock no.	Percent smutted heads
15.	Henry x Cadet	N. Dak.	C.I. 12966	67
16.	Lee	Minn.	C.I. 12488	67
17.	Lee selection 1952 Brawley	Minn.	549	65
18.	K58 x Newthatch	Minn.	C.I. 13032	64
19.	Rushmore ^{2}X Surpresa	N. Dak.	C.I. 12972	58
20.	Timstein x Newthatch	Minn.	II-42-22	57
21.	Y-Kt 2254-2c-1y-1c	Mexico		57
22.	Lee	Minn.	C.I. 12488	55
23.	Mida	N. Dak.	C.I. 12008	54
24.	Frontana x Mida	Minn.	II-46-117	54
25.	Kt-Y 2587-1T-1H-1T	Mexico		54
26.	Mida x Lee	Minn.	II-46-122	54
27.	Kentana 51B	Mexico		53
28.	Yaqui	Mexico		53
29.	(A-K) (M-S) 1088-2y-5c-5c-1c	Mexico		53
30.	Kt-Y (May segregate) 2585-3y-4c	Mexico		52
31.	Lee	Minn.	C.I. 12488	52
32.	Kenya-Gular-Pilot X K58-Newthatch	Min..	II-44-58	51
33.	Frontana X II-44-29	Braw. 801 Minn.	II-50-36	49
34.	T-K^2 775-1c-1y-2c	Mexico		48
35.	Frontana X II-44-29	Minn.	II-50-25	48
36.	K 338AC2E2	Kenya	C.I. 12880	46
37.	Frontana X II-44-29	Minn.	II-50-34	46
38.	Pilot ^{2}X Regent	Mont.	C.I. 13042	46
39.	Mida	N. Dak.	C.I. 12008	44

Table 1. (continued).

No.	Variety or cross	Source	C.I. no. or nursery stock no.	Percent smutted heads
40.	Henry x Cadet	N. Dak.	C.I. 12779	43
41.	Ceres	N. Dak.	C.I. 6900	42
42.	Mida x Kenya 117A	Minn.	C.I. 12964	41
43.	Marquis	Canada	C.I. 3641	40
44.	Mida	N. Dak.	C.I. 12008	40
45.	(K-T) My 2156-6c-2y-1c	Mexico		39
46.	Mida	N. Dak.	C.I. 12008	38
47.	Frontana x Thatcher	Minn.	II-46-5	37
48.	Timstein x Newthatch	Minn.	II-42-22	36
49.	Mida	N. Dak.	C.I. 12008	36
50.	Kentana	Mexico		36
51.	Henry	Wisconsin	C.I. 12265	34
52.	Kenya-Gular-Pilot X K58-Newthatch	Minn.	II-44-52	32
53.	Red Egyptian X Frontana	Minn.	C.I. 13048	31
54.	Lee x Frontana	Minn.	C.I. 12957	31
55.	Rushmore	S. Dak.	C.I. 12273	30
56.	(K-M^2) Me 1442-1c-1c-1c-1c	Mexico		29
57.	(N-M) (Mt-K) 908-7c 4c-1c-1y	Mexico		27
58.	Frontana X II-44-29	Minn.	II-50-35	26
59.	Frontana X II-44-29	Minn.	II-50-17	22
60.	Frontana	Brazil	C.I. 12470	21
61.	K58	Kenya	C.I. 12471	20
62.	Red Egyptian x Frontana	Minn.	C.I. 13048	19
63.	Mayo 52	Mexico		14
64.	K117A	Kenya	C.I. 13140	12
65.	Rival	N. Dak.	C.I. 11708	8

Table 1. (continued).

No.	Variety or cross	Source	C.I. no. or nursery stock no.	Percent smutted heads
66.	Rushmore^{2}X Surpresa	S. Dak.	C.I. 12973	8
67.	1520 X 1752	Mont.	C.I. 13041	6
68.	Willet	Minn.	C.I. 13099	6
69.	Frontana X Thatcher	Minn.	II-47-37	6
70.	Selkirk	Canada	C.I. 13100	6
71.	Thatcher X Surpresa	Minn.	C.I. 12641	5
72.	Me^2-N 1930-17c-3T-1H	Mexico		5
73.	Taboado #5	Mexico		3
74.	Frontana X Thatcher	Minn.	II-46-14	2
75.	Thatcher	Minn.	C.I. 10003	2
76.	Red Egyptian X Mida	Minn.	II-44-9	2
77.	Rival X H49.4.3.3-1	Minn.	II-39-88	2
78.	Selkirk	Canada	C.I. 13100	1
79.	Thatcher	Minn.	C.I. 10003	1
80.	Lee X Mida Sib.	N. Dak.	C.I. 13033	0
81.	Lee X Mida Sib.	N. Dak.	C.I. 13034	0
82.	Lee X Mida Sib.	N. Dak.	C.I. 13035	0
83.	Lee X Mida Sib.	N. Dak.	C.I. 13036	0
84.	Lee X Mida Sib.	N. Dak.	C.I. 13037	0
85.	Lee X Mida Sib.	N. Dak.	C.I. 13038	0
86.	Lee X Mida Sib.	N. Dak.	C.I. 13039	0
87.	Henry X Thatcher-W38-111.1-Hope	Wis.	C.I. 13098	0
88.	Frontana X Thatcher	Minn.	C.I. 13030	0
89.	Frontana X Thatcher	Minn.	C.I. 13031	0
90.	Frontana X Thatcher	Minn.	C.I. 13046	0
91.	Moen wheat (Thatcher selection)	Minn.	I-53-1	0
92.	K58 X Mida-Newthatch	Minn.	II-45-27	0

Table 1. (continued).

No.	Variety or cross	Source	C.I. no. or nursery stock no.	Percent smutted heads
93.	Newthatch X Frontana	Minn.	II-46-96	0
94.	K58 X Mida-Newthatch	Minn.	II-45-29	0
95.	Kenya-Gular-Pilot X K58-Newthatch	Minn.	II-44-48	0
96.	Mida-Newthatch X K117 A	Minn.	II-45-23	0
97.	Hope	S. Dak.	C.I. 8178	0
98.	Newthatch	Minn.	C.I. 12318	0
99.	Me^2-N 1930-17c-2T-9H	Mexico		0
100.	K-U 1122-4c-1c-1c-3y-3c-1y	Mexico		0
101.	Thatcher	Minn.	C.I. 10003	0
102.	Frontana X Thatcher	Minn.	C.I. 13030	0
103.	Willet	Minn.	C.I. 13099	0
104.	Redman	Canada	C.I. 12638	0
105.	Mindum	Minn.	C.I. 5296	0
106.	Carleton	N. Dak.	C.I. 12064	0
107.	Stewart	N. Dak.	C.I. 12066	0
108.	Nugget	N. Dak.	C.I. 12620	0
109.	Sentry	N. Dak.	C.I. 13102	0

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TRENDS IN DISEASE LOSSES OF WHEAT, OATS, AND BARLEY IN NORTH CAROLINA FROM 1950 TO 1954

J. G. Moseman, J. C. Wells, and T. T. Hebert[1]

More than 800,000 acres of wheat, oats, and barley were harvested annually in North Carolina during the last decade. Approximately 400,000 acres each of wheat and oats and about 40,000 acres of barley were harvested. Wheat, oats, and barley yielded 22, 37.5, and 34 bushels per acre, respectively, in 1954.

The percentage decrease in the potential yield of these three grains in North Carolina from various diseases was estimated for each of the last five growing seasons (Table 1). Although the loss estimates may be somewhat inaccurate, the year to year trends for the various diseases are thought to be significant.

WHEAT LOSSES

The decrease in severity of leaf rust[2] following the 1949-50 season probably was due to the increased growing of resistant varieties derived from the crosses with the two Brazilian wheats, Frondoso and Fronteira, and to weather conditions less favorable for the development of leaf rust in some years. Derivatives of these two varieties, with the exception of Atlas 50, have exhibited exceptionally good resistance to leaf rust. The increased acreage of these rust resistant varieties in South Carolina, southern Georgia, and Florida has aided in checking a build-up of leaf rust inoculum. In 1954 a new race or races of leaf rust found in eastern North Carolina and in Georgia attacked some of the derivatives from the Brazilian wheats, but not more than a trace of leaf rust was observed on Atlas 66.

The decrease in loss from powdery mildew during the 1950-51 and 1951-52 seasons likely was due to the release of the Atlas varieties, and to unfavorable weather conditions for the pathogen. The Atlas and other varieities derived from the Brazilian wheats are not resistant to the races of powdery mildew now present in North Carolina. Large losses may occur should weather conditions become favorable for the development of the organism.

The losses from glume blotch and scab have fluctuated with the prevailing weather conditions which have favored or hindered the development of the pathogens.

The soil-borne viruses (Marmor tritici var. typicum and M. tritici var. fulvum) were not generally recognized as a limiting factor in wheat production in North Carolina until the release of the Atlas varieties and other Brazilian-derived wheats, although some of the older varieties were known to be susceptible to the viruses. When the farmers began to grow these new varieties they increased the amount of nitrogen top-dressing which accentuated field symptoms of the virus diseases. The new varieties Anderson and Taylor are more tolerant to the viruses than are most varieties grown at the present time. Some highly resistant selections from the variety Taylor are being increased for release to growers.

The insignificant losses resulting from loose smut are difficult to explain. The varieties now being grown are susceptible to the races identified by Bever[3] from this area. Weather conditions prevailing at the stage the wheat is susceptible to infection may be unfavorable for the development of the disease in this region.

OATS

The decrease in yields of oats resulting from plant pathogens has not been very large since the 1949-50 growing season. Losses from leaf blotch have decreased appreciably. The weather in the 1949-50 season was very favorable for the development of leaf blotch. The increase in the acreage of Arlington and Victorgrain 48-93 varieties may have helped to reduce the loss from H. avenae.

Losses from other Helminthosporium species vary, depending on the growing season, ro-

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[2] Names of pathogens given in Table 1.

[3] Results from cooperative testing and correspondence with Dr. W. M. Bever, pathologist (U. S. D. A.), at University of Illinois, Urbana, Illinois.

Table 1. Estimated decrease in yield of small grains in North Carolina resulting from diseases in 1949-50, 1950-51, 1951-52, 1952-53, and 1953-54, in percent of potential yield.

CROP Disease	Pathogen	Percent decrease in yield				
		1949-50	1950-51	1951-52	1952-53	1953-54
WHEAT						
Leaf rust	Puccinia rubigo-vera var. tritici	10	2	4	3	4
Powdery mildew	Erysiphe graminis	10	1	1.5	1	1
Glume blotch	Septoria nodorum	3.5	t	1.5	2	3
Scab	Fusarium spp. and Gibberella zeae	2	1	2	1	2
Loose smut	Ustilago tritici	1.5	1	t	t	t
Mosaic	Marmor tritici var. typicum					
	Marmor tritici var. fulvum	t	2	3	2	2
Total		27	7	12	9	12
OATS						
Crown rust	Puccinia coronata	-	-	-	-	-
Leaf blotch	Helminthosporium avenae	10	3	3	3	2
Helminthosporium spp.	Helminthosporium spp.	2	2	2	1	1
Smut	Ustilago avenae and Ustilago kolleri	2	1	1	1	1
Mosaic	Marmor terrestre var. typicum					
	Marmor terrestre var. oculatum	1	t	t	t	t
Total		15	6	6	5	4
BARLEY						
Leaf rust	Puccinia hordei	10	1	t	1	t
Powdery mildew	Erysiphe graminis	5	t	1	1	1
Helminthosporium spp.	Helminthosporium spp.	5	1	1	1	1
Scald	Rhynchosporium secalis	2	t	t	t	10
Scab	Fusarium spp. and Gibberella zeae	5	1	2	1	2
Loose smut	Ustilago nuda	10	10	10	8	5
Total		24	13	14	12	19

tation practices followed, and the amount of oats sown early for pasture. When oats are sown in late August or early September for pasture on land previously in oats, the losses from certain Helminthosporium species are often very large.

Although the smuts can be controlled by the standard recommended mercury seed treatments, they have been observed in numerous fields every year.

Except for the year 1949-50, when some losses were observed from the soil-borne viruses the losses from these viruses and from crown rust have been insignificant. Since most of the varieties grown in the State are resistant to these pathogens, losses from them would not be expected.

BARLEY

The severe loss from leaf rust and powdery mildew in the 1949-50 growing season probably was due to the favorable weather conditions. The two most widely grown varieties, Colonial and Calhoun, are susceptible to both these diseases but usually do not become heavily infected until after heading. The Davie variety, that was released to the farmers in the fall of 1953, has mature plant resistance to leaf rust and to most races of powdery mildew.

Losses from Helminthosporium have been light. If more barley were sown in late August or early September for pasture, losses from spot blotch (H. sativum) would increase rapidly, especially where crop rotation is not followed.

Scab caused more loss in 1949-50 than in the other years. This likely was due to the damp, warm weather from the time the grain headed until it was harvested. The weather has been relatively dry during the last four spring seasons.

Scald is considered a serious disease in the winter barley area, but occurs only sporadically in North Carolina. However, the Colonial variety was damaged severely by scald during the 1953-54 growing season. Losses up to 50 percent were observed in some fields. The disease was most severe in fields that had been in barley the previous year. Davie apparently is resistant to scald as it has stood up well to date.

Following the 1949-50 and 1950-51 growing seasons, when the estimated decrease in yield of barley resulting from loose smut was 10 percent of the potential yield, a hot-water seed-treatment program was started in cooperation with county agents and extension specialists. Selected farmers were requested to bring about 10 bushels of seed to a designated location. The county agent and the extension specialist supervised the hot-water treatment of this seed. The program was started in four counties in the fall of 1951. The amount of seed treated increased from 156 bushels in 1951 to 253 bushels and 850 bushels in 1952 and 1953, respectively. Twelve counties cooperated in 1954.

The results from this treatment have been encouraging. The yields obtained by farmers who sowed the treated seed have been good. Loose smut control has been virtually 100 percent and losses resulting from loose smut have decreased in the cooperating counties. Until a variety resistant to loose smut is released, it will be necessary to continue hot-water seed treatment.

FIELD CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, U. S. DEPARTMENT OF AGRICULTURE IN COOPERATION WITH THE NORTH CAROLINA AGRICULTURAL EXPERIMENT STATION, RALEIGH, NORTH CAROLINA

DELETERIOUS EFFECTS OF DRYING ON SURVIVAL OF BROOMRAPE SEED IN SOIL

Stephen Wilhelm[1]

Orobanche ramosa L., the branched broomrape, an angiosperm parasitic on several important crops, is troublesome on tomatoes in a few fields in Alameda County of California. Its distribution and host range, which includes several weeds, are given in recent publications (4, 8).

The apparent capacity of the broomrape seed to lie dormant in soils for long periods, and of the parasite to recur in profusion in response to planting a favorable host such as tomato, is well established by field observations (5, 6, 8). Spread in California has been accounted for satisfactorily by flood waters and by transporation of seed-infested soil on farm implements. Wind dissemination appears to have been of no importance, notwithstanding the fact that the seeds are small and are produced in abundance.

FIGURE 1. The branched broomrape, Orobanche ramosa L. approaching maturity in an 8-inch pot of field infested soil. Note the many seed capsules. The tomato plants, needed to stimulate germination of the seed and to support the pest, have been cut off.

No condition seriously adverse to seed survival seems to have been discovered. Though records seem not to be available, the possibility is good that broomrape was introduced into California as well as into the United States (5) as a contaminant of hemp seed. According to Dewey (3), broomrape seed may become stuck to the gummy flower parts of hemp if the hemp seed crop falls to the ground in harvesting. It seems unlikely that hemp was ever grown in Alameda County of California as a fiber crop, but records indicate that it was grown for fiber in parts of the lower Sacramento Valley, in Solano, Kern, and possibly Stanislaus Counties, and extensively in the Imperial Valley and parts of Arizona (1, 2, 3, 7). Its desirability as an orna-

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Table 1. Effects of drying on the survival of broomrape seed in field soil as indicated by the number of shoots appearing per unit volume of soil in response to tomato plants.

Soil sample*	Number of shoots of broomrape appearing per unit volume of undried and dried soil		
	Undried	Dried three months	Dried six months
1	10	0	0
2	39	0	0
3	19	3	0
4	1	0	0
Totals	69	3	0

*Each sample consisted of five 8-inch pots of field soil naturally contaminated with broomrape seed.

mental and annual shade tree, especially for chicken yards, and the fancy of many nurserymen to grow unusual plants, may have promoted the distribution of hemp still more widely. Kentucky was the source of some, perhaps most, of the hemp seed planted commercially in California (1, 3) at a time when broomrape was a serious pest in the former State. The fact that broomrape is apparently unknown today in the dry warm areas of California mentioned above, where hemp culture flourished 30 to 40 years ago, together with lack of evidence of successful wind dissemination of the seed either in the coastal areas now infested or in Kentucky (6), suggested that seed of this parasite may succumb to prolonged drying.

The only method known at present whereby broomrape seed can be detected in soil is to plant a preferred host such as tomato or tobacco. In the presence of these plants, broomrape seed germinates, the seedling parasitizes the host roots, and within two to three months flowering shoots appear above the ground (Fig. 1). Earlier studies showed that both tobacco and tomato plants would stimulate germination of broomrape seed.

To determine the effects of drying on the survival of broomrape seed, soil samples were collected from a field which in the year previous showed an abundance of the parasite. The soil was divided into three parts and put into 8-inch pots. One part was tested for the presence of viable broomrape seed by planting tomato immediately, and the other parts after drying for three and six months respectively. Drying was at indoor temperatures ranging from 60° to 85° F. Each sample (Table 1) consisted of five 8-inch pots of the soil.

After the experimental drying, five Pearson tomato seedlings were transplanted into each 8-inch pot of soil and a record was kept through 3 1/2 months, of the number of broomrape shoots that appeared. The number of broomrape shoots appearing per unit volume of soil, such as that of an 8-inch pot, is thought to be indicative of the seed density in the soil (9) (Table 1).

Soil dried for three months under the conditions of this experiment showed a striking reduction in the number of broomrape shoots appearing, and after six months of drying, no broomrape appeared. These data suggest that the seed had succumbed during drying. Admittedly these data are in sharp contrast to Garman's (6) who reported germination of broomrape in soil kept in pots for 13 years, and no reasons can be offered at present for the discrepancy.

In fields in coastal California in which only non-hosts or weeds were present, broomrape has survived up to four and five years, but marginal perpetuation on little preferred weed hosts may have augmented this survival. Pot tests never simulate exactly the conditions in the field, but the absence of the pest from warmer and drier parts of California may support the theory that long periods of drying kill broomrape seed in the soil.

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UNIVERSITY OF CALIFORNIA, BERKELEY

1954 PLANT DISEASE SURVEY FOR NEW MEXICO

Philip J. Leyendecker

1. Alfalfa mosaic (Marmor medicaginis Holmes) was positively identified as occurring on the following legumes: Trifolium repens L. (Ladino); Melilotus officinalis (L.) Lam., and Medicago sativa L. Mosaic was first discovered in a breeding block of alfalfa on the New Mexico Experiment Station farm at State College. The infection was so severe that considerable differences in the degree of susceptibility could be measured. Under field conditions large leaflets and thick stems seemed to be correlated with susceptibility. A field survey revealed further that the disease was also present in commercial plantings in the Pecos and Mesilla Valleys.

2. Curly top (Ruga verrucosans Carsner & Bennett) was general over the State causing considerable damage to tomato, red pepper, and beans. Extreme disease expression was noted in the Portales area on snap beans, tomato and red pepper, where populations of the sugarbeet leafhopper (Circulifer tenellus Baker) were found to be unusually high. Numerous commercial fields suffered from 25 to 50 percent loss because of the disease.

3. Below is a short supplement to Table 1 which appeared in PDR 37: 552-553. 1953. The ornamentals were grown in the same garden and under the same conditions as in 1953.

Table 1. Reaction of ornamental flowering plants to the curly-top virus, Ruga verrucosans, under natural conditions of infection in Southern New Mexico (1954).

FAMILY Scientific name	Name of plant: Common	Variety	Curly-top reaction[a]
CARYOPHYLLACEAE			
Lychnis coronaria	Mullein pink	Atrosanguinea	**
COMPOSITAE			
Aster sp.	Aster	Early Burpeeana	*
PLUMBAGINACEAE			
Armeria pseudoarmeria	Sea pink	Hybrids	0
RANUNCULACEAE			
Delphinium sp.	Larkspur	Pacific Giants	**
ROSACEAE			
Geum chiloense	Avens	Mrs. Bradshaw	*
Geum chiloense	Avens	Lady Stratheden	*
SCROPHULARIACEAE			
Penstemon gloxinioides	Beard tongue	Hybrids	0
VALERIANACEAE			
Valeriana officinalis	Garden-heliotrope		**

[a] 0 = Resistant.
* Susceptible.
** Highly susceptible.

4. Bean rust (Uromyces phaseoli (Pers.) Wint. var. typica Arth.) has been rather sporadic in its appearance in New Mexico for the last decade. However, in the last few years it has been increasing in severity because of the increased acreage of dried beans grown under

irrigation. This year rust was very severe in the irrigated Estancia and Mimbres Valleys. Conservative estimates made by growers indicated that 45 to 50 percent of the crop was destroyed, which amounted to approximately a $500,000 loss. Dusting with sulfur was not effective in controlling the pathogen since it was applied late in the season after the rust had almost completely defoliated the plants.

5. Big bud (Chlorogenus australiensis Holmes), which was reported from New Mexico the first time in 1950, was very prevalent in the Mesilla Valley on tomato and red pepper. In some tomato fields (J. Moran variety) 2 to 3 percent of the plants were infected. Very few infected plants produced salable tomatoes. Fewer red pepper plants showed big-bud symptoms. However, fruit symptoms which had not been seen before were noted when the older flowers set pods which matured and colored very unevenly, forming irregular red and green mosaic patterns on the fruit. Since the discovery of its presence in 1950, the virus seems to be increasing in incidence.

6. Verticillium wilt (Verticillium albo-atrum Reinke & Berth.), a very serious disease on Upland cotton in New Mexico, was found for the first time causing extreme damage on eggplant and okra. Large commercial plantings in the Mesilla Valley suffered from 50 to 90 percent loss in production. Verticillium was also found infecting eggplant under field conditions 250 miles to the north at Bernalillo, New Mexico. A number of weed hosts of Verticillium in New Mexico which have not been reported previously are: Tribulus terrestris L. (goathead), Xanthium commune Britton (cocklebur), and Sphaeralcea angustifolia (Cav.) G. Don. (copper mallow).

7. Southern blight (Sclerotium rolfsii Sacc.) caused only slight damage on peanuts as compared with previous years in the Portales area. Disease expression was late and the yield was not so markedly reduced as in previous years. A field of snap beans at Portales was found in which 5 to 10 percent plant mortality occurred because of southern blight. This was the first report of Sclerotium causing losses on snap beans in the area. Southern blight was also found to be responsible for a 25 to 30 percent loss of mature plants in a commercial seed field of Zinnia elegans Jacq. at Garfield, New Mexico. The damage followed heavy rains where runoff water stood in low areas of the field.

8. Mosaic (Marmor cucumeris Holmes) was almost universally found on chile (Capsicum annuum L.) plantings in northern New Mexico. Leaf mottling was so severe in some instances that the plants were stunted and the green pods were covered with irregular brownish raised areas. Many growers reported that they have not seen the disease before. Should the virus appear in such proportions next year, definite control measures will be necessary.

9. Fusarium wilt (? Fusarium annuum Leon.) is the most prevalent disease of red pepper in New Mexico. It is found in varying degrees in every red pepper planting in the State. Plantings range in size from a few rows in a garden to large commercial acreages. Many fields have been abandoned for chile production because of the malady. This year the loss was 35 to 40 percent of the crop. Preliminary studies have indicated that there is some question as to the organism responsible for chile wilt in New Mexico. It is believed that Phytophthora capsici Leon. is responsible for the initial root infection and damage and Fusarium annuum appears to be secondary. The Fusarium is readily isolated from diseased material, but Phytophthora capsici requires special handling to make isolations before it is completely overgrown by Fusarium. Greenhouse tests have failed to show that the Fusarium isolated[1] from diseased red pepper plants is pathogenic. On the other hand, P. capsici under identical greenhouse conditions caused complete collapse of the root bark, a symptom commonly found in the field and heretofore attributed to F. annuum.

10. Black root rot (Thielaviopsis basicola (Berk. & Br.) Ferr.) has been previously reported from New Mexico on long staple cotton (Pima 32). This year the disease was discovered in a commercial planting of upland cotton (1517C) near Berino, New Mexico. Fifty to 60 percent of the plants in two areas, 60 to 70 feet across, were killed by black root rot. Inspection of the roots showed the typical swelling and internal browning of the central woody cylinder. The organism was associated with nematode galls, as was the case in long staple cotton.

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[1] Isolations made by Dr. W. C. Snyder, University of California, Berkeley.

11. Southern anthracnose of alfalfa (Colletotrichum graminicola (Ces.) G. W. Wils.) appeared in a number of commercial fields in the Mesilla and Pecos Valleys. In some fields the percentage of dead stems resulting from anthracnose infection ran as high as 20 to 30 percent. These fields appeared to be suffering from drought, but examination of the stems revealed that they had been girdled by the pathogen. The incidence and severity of the disease appeared to be on the increase. Seed fields were especially hard hit, seed yields being reduced as much as 20 percent.

12. Rosette (zinc deficiency) of pecan is becoming more and more common in southern New Mexico since the pecan is being planted as a shade tree on light sandy alkaline soil. Many young trees planted in the last few years show typical rosette growth characteristic of zinc deficiency. In some cases the condition can be corrected by the annual application of zinc sulfate to the soil.

13. Rasp leaf, a virus disease of sweet cherry, was positively identified from Mountain Park, New Mexico, this year[2]. Local orchardists believe that the disease has been in the area for a number of years. It occurs on large producing trees and there is some evidence that the virus has been transmitted from tree to tree by an insect vector.

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[2] Paul C. Duffield and Philip J. Leyendecker. 1954. Rasp leaf virus disease discovered in New Mexico on sweet cherry. Plant Dis. Reptr. 38: 832-833.

UNUSUAL RECORDS OF PLANT DISEASES

DOWNY MILDEW OF CORN IN TEXAS

By Marvin D. Whitehead

A 100 percent infection with downy mildew of a ten-acre, creek-bottom field of Texas Hybrid 30 corn was observed in August 1954 at De Leon, in north-central Texas. This field, that in past years has produced 100 to 125 bushels of corn per acre, was completely destroyed by this disease in 1954. When the corn was up to one-half full stand, the field was flooded for about ten hours. The temperature was near freezing during this period. After the overflow water receded, the corn came up to a full stand and appeared healthy for a short time. The farmer noted chlorotic streaks on the leaves when the plants were about two feet high, and the plants appeared to lack vigor for the rest of the season. Approximately one-half of the plants failed to produce tassels and most of the tassels that did form were grotesquely malformed. Foliate tassels 12 inches in diameter and 16 inches long were common.

Oospores of a downy mildew were found in all plants observed. In a high percentage of the mature plants the tissues adjoining the vascular bundles of all the foliar portions of the tassels, ear husks, leaves, and leaf sheaths were heavily filled with oospores. No sporangium formation was noted, but from oospore morphology and host symptoms the downy mildew is tentatively considered to be Sclerophthora macrospora (Sacc.) Thirum. et al. (Sclerospora macrospora Sacc.).

Minor infections were observed at College Station and Hufsmith in South Central and South Texas during 1952 and 1953.

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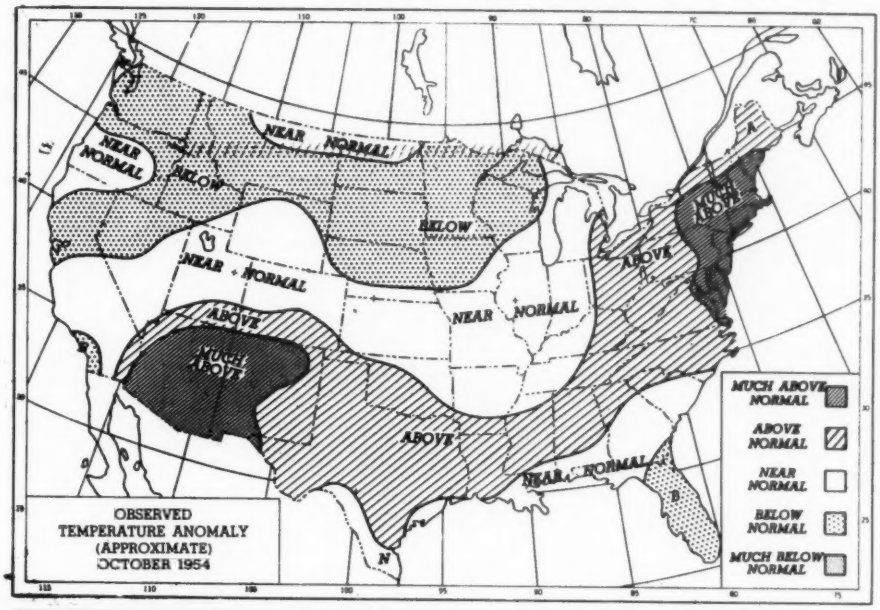

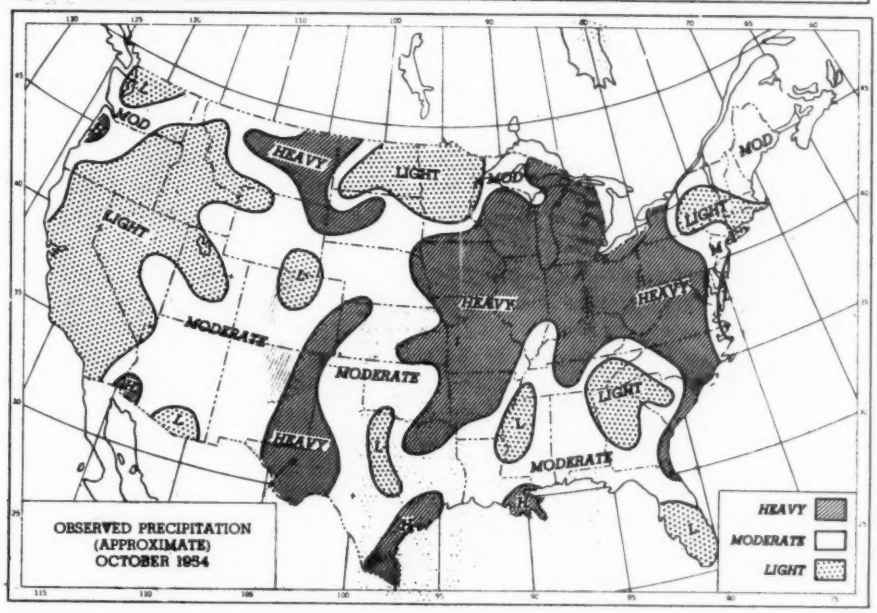

The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.

MANUSCRIPTS FOR THE PLANT DISEASE REPORTER

(See also PDR 35: 121-122; 36: 214, 494)

(1) GENERAL: The Reporter page measures 9 inches long with the heading or 8 3/4 inches for the text part, by 6 inches wide. The copy is typed on a larger page, 11 1/4 inches of text or 12 inches overall in length by 8 inches in width, and reduced 25 percent in the photographic process of reproduction. Illustrations or tables larger in either dimension will take a correspondingly greater reduction. Only one size of type is available for text, footnotes, or tables.

(2) MANUSCRIPTS should be the original ribbon copy, not carbons, clearly typed and double-spaced throughout, including tables, footnotes, and bibliographies. (Note -- only one copy is needed.) Footnotes should be typed at the bottom of the page.

(3) ABSTRACTS are requested for all except very short articles. (PDR 38: 316).

(4) CAUSES OF DISEASES should be named. For bacteria, fungi, nematodes, etc., give the Latin name of the organism; for viruses either or both the accepted common name of the virus or a Latin name if you prefer it and there is one; for non-parasitic diseases state the causal factor if it is known. If the cause of a disease has not been determined say so. (PDR 36: 494).

(5) LITERATURE REFERENCES should be given in alphabetical order and numbered for citation in the text. We follow the AIBS suggestion of placing the year of publication after the author's name. Please check your titles carefully since we cannot do it for you. Be sure that text citations and bibliography agree; that foreign-language references are correct; that number or month is cited for periodicals that are not paged consecutively throughout the volume.

(6) NAMES OF FUNGICIDES should be given according to the suggestions that will be published in Phytopathology before long, and later repeated in the Reporter. In the meantime see PDR 35: 122.

(7) ILLUSTRATIONS should be sent to us unmounted. To prevent mistakes, write figure numbers on the back, and mark the top of each print when necessary. A sketch can show a preferred arrangement but please keep in mind page size, shape, and standard reduction (see above under General), and remember that figure titles and legends are part of the page. Lettering should be clear and large enough to be legible after reducing. Drawings, maps, and graphs can be photographs or originals, but should be finished and ready for reproduction, not just sketches.

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(11) We are the official correspondents in the United States of the PLANT PROTECTION BULLETIN published monthly by the United Nations Food and Agriculture Organization. We would appreciate it if plant pathologists would send us suitable material from time to time for inclusion in our monthly summary reports. Please mark it clearly so that we will know it is intended for the Bulletin. (See PDR 36: 492; 37: 637) -- P. R. M.